

DIAGNOSIS AND TREATMENT OF
ARTHRITIS AND ALLIED DISORDERS

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BY

H. M. MARGOLIS, M.D., M.S. (in med.), F.A.C.P.

Chief, Arthritis Service, St Margaret Memorial Hospital

Associate in Medicine, Montefiore Hospital

Consultant in Medicine, Pittsburgh Diagnostic Clinic

WITH 140 ILLUSTRATIONS



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DIAGNOSIS AND TREATMENT OF ARTHRITIS AND ALLIED DISORDERS

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*To the memory of my mother
and to my wife*

drawn extensively from scientific investigations and reports of many observers. Specific references in the text to the many original sources of medical literature from which valuable data have been taken are avoided in order to simplify the presentation of the subject. The more important bibliographic references especially those in journals most likely to be readily available to the practitioner are listed at the end of each chapter. In addition the concluding section of the book contains a list of books and monographs dealing with the broader aspects of rheumatic disease, which may be used for further reference. It is hoped that the careful selection of the bibliography will add to its usefulness.

Important newer concepts concerning etiology, pathogenesis and treatment—including the controversial aspects of focal infection, the relation of intervertebral disk protrusion to the pathogenesis of low back and sciatic pain, the present status of chrysotherapy in atrophic arthritis and of therapy with sulfanilamide in gonococcal arthritis—have been critically appraised.

A practical approach to the subject has been maintained throughout, with an aim toward simplicity both in the organization and presentation of the subject matter. The space allotted to the various topics discussed is based largely on their importance to the practicing physician. Therefore, the more common forms of rheumatic disease are given more detailed consideration than those which the physician encounters only rarely. In the latter case references for further study are provided in the bibliography. For the same reason practical diagnostic and laboratory procedures are discussed more fully than the more complicated and frequently less useful ones that belong more in the research clinic or laboratory. Those diagnostic measures which the physician may readily employ in his own office have been described in greatest detail, the actual technique of the procedure in such cases usually being indicated.

Since clinical appraisal of any disease cannot be separated from an appraisal of the patient and his environment, his constitutional make up and the socio-economic setting in which the disease occurs, discussion of these aspects of the subject—an integral part, I believe, of any clinical consideration of rheumatic disease—has been included.

I realize that the inclusion of certain topics is not orthodox. Discussions of low back pain, radicular pain and the like have usually not been presented with discussions on arthritis. But then, I am convinced that many phases of rheumatic disease exclusive of the arthritides, but closely related to them by symptomatology, must not only frequently be considered in differential diagnosis but may furthermore be treated successfully by the physician.

In general the viewpoint expressed was substantiated by personal experience. Opinions of others have not been disregarded, however. On impor-

tant controversial topics especially, such divergent views are stated, but when my own experience seemed conclusive enough, I have not evaded saying so

Every important phase of treatment has been discussed, for the management of the patient with rheumatic disease must be an integrated performance, diligently supervised at every turn by the physician in charge. There can be no prefabricated program of treatment for each patient, there is so much variation in the clinical picture and in the requirements of individual cases. I have attempted, however, to discuss the fundamentals of treatment and to evaluate their relative merits and deficiencies. Above all, the aim has been to stress procedures which are useful and applicable in practice. The simplest, yet most effective way of achieving a desired result is always given preference to the more elaborate, more dramatic therapeutic setting. Useful, inexpensive measures applicable in the home treatment of such cases are emphasized, especially in relation to physiotherapeutic management. In this way, it is hoped, the book will constitute a practical guide designed to meet the requirements of bedside practice.

I have purposely refrained from detailed discussion of many remedies based on the flimsiest of evidence, that are constantly being "tried and heard about" in the treatment of arthritis. It is hoped that the reader will not regard this as a serious omission. Nor did it appear worth while to clutter the text with detailed description of therapeutic procedures which, though highly popular are in my estimation impractical, ineffective, or dangerous, either such procedures are mentioned briefly, or mention of them is altogether omitted. Reference to such topics is, however, included in the bibliography, so that the reader may have access to detailed discussion, if he is interested.

Practical experience shows that deformity is not an inevitable phase of arthritic disease, but rather a complication which may be averted by timely, adequate management. Furthermore, I regard that phase of therapy aiming at prevention and correction of deformities in arthritis as an integral part of the general therapeutic program, a phase of therapy all too often delegated disinterestedly to the orthopedist. As a matter of fact, in most instances, especially in early cases, the physician may carry out, unaided practically all of the requisite steps of simple orthopedic management, and thus prevent the deformities we dread so much. Therefore I have provided in Chapter xv a detailed, though simple outline of the factors that are responsible for such deformities and of the means which may be employed for their prevention and correction. To emphasize graphically this especially important but badly neglected phase of this subject, this chapter is profusely illustrated so that recognition of the wide range and practical simplicity of the measures to be employed may be greatly facilitated.

Since I believe too that even purely surgical phases of management applicable to cases of rheumatic disease should be clearly understood by the attending physician the rationale and indications of such therapy have been outlined. Details of the surgical procedures to be employed have, for the most part, been omitted because it was not the intention to supply a technical guide for the surgeon. The aim was rather to indicate to the practitioner the general scope and magnitude of surgical procedures which may be recommended by a consultant.

To many who contributed to making this book possible I am deeply grateful. Foremost among these is my former chief and teacher, Dr. Philip S. Hench, who first stimulated my interest in the rheumatic diseases. I also wish to pay tribute and acknowledge an unbounded sense of debt to the late Drs. William and Charles Mayo, who made possible the Clinic where I was privileged to spend several of the happiest years, where I studied and found myself.

I owe thanks to many others who helped so willingly to Mrs. T. D. Howe for the discerning care with which she read and criticized the manuscript; to Dr. V. W. Eisenstein for help on the sections dealing with the neurologic counterfeits of rheumatic disease; to Dr. Eben W. Fiske and Dr. A. S. Browdie for suggestions and help on the orthopedic aspects of the subject; to Mr. A. Levin for painstaking care in making the photographs with which the text is illustrated; to Miss E. M. Shackelford for the drawing she contributed; and to Miss Ruth Clarke for aid in checking proof.

To my secretary, Mrs. Elsie D. Steinmann, I am grateful for the faithfulness with which she earned the additional work imposed upon her, and to Miss Selma Haendler for careful stenographic assistance. The efficient help of Miss Frances Zewe and Miss Marguerite Lawson in the care of our patients afforded many extra hours which could be devoted to this task.

I thank various writers for permission to reproduce a number of worthwhile illustrations, and Dr. E. A. Codman and Dr. Roy R. Snowden, Director of the Pittsburgh Diagnostic Clinic, for lending me several plates for illustrations.

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Without the encouragement and help of my wife this book could not have been written. And to my son, Richard, I want to express my admiration for the graciousness with which he bore his sacrifice of many hours of play in which we could not indulge when we both wished so much to do so.

H. M. MARGOLIS

Pittsburgh, Pennsylvania

CONTENTS

PREFACE	VII
INTRODUCTION	1

Part I—Atrophic Arthritis

I CLASSIFICATION OF THE ARTHRITIDES	17
II ATROPHIC ARTHRITIS SOME BASIC CONSIDERATIONS	20
III THE PRECIPITATING ETIOLOGIC FACTOR IN ATROPHIC ARTHRITIS	35
IV PATHOLOGY OF ATROPHIC ARTHRITIS	45
V PATHOGENESIS OF ATROPHIC ARTHRITIS	56
VI FOCAL INFECTION IN ATROPHIC ARTHRITIS	65
VII THE VARIOUS FOCI OF INFECTION IN ARTHRITIS	74
VIII ATROPHIC ARTHRITIS THE CLINICAL MANIFESTATIONS AND DIAGNOSIS	81
IX ATROPHIC ARTHRITIS OF THE SPINE	113
X STILL'S DISEASE	121
XI PSORIATIC ARTHRITIS	125
XII FOCAL ARTHRITIS	127
XIII THE TREATMENT OF ATROPHIC ARTHRITIS	129

Part II

XIV THE PREVENTION AND CORRECTION OF DEFORMITIES IN CHRONIC ARTHRITIS	167
XV AN ILLUSTRATED GUIDE TO THE RECOGNITION OF THE CHARACTER AND CAUSES OF ARTHRITIC DEFORMITIES AND OF MEANS FOR THEIR PREVENTION AND CORRECTION	183
XVI PHYSICAL THERAPY IN ARTHRITIS	231

Part III—Hypertrophic Arthritis

XVII HYPERTROPHIC ARTHRITIS SOME GENERAL CONSIDERATIONS	251
XVIII PATHOLOGY OF HYPERTROPHIC ARTHRITIS	259
XIX CLINICAL MANIFESTATIONS OF HYPERTROPHIC ARTHRITIS	264
XX TREATMENT OF HYPERTROPHIC ARTHRITIS	279

Part IV—Rheumatic Fever

XXI RHEUMATIC FEVER	291
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Part V—Specific Arthritides

XXII GOUTY ARTHRITIS	311
XXIII GONOCOCCAL ARTHRITIS	326
XXIV TUBERCULOUS ARTHRITIS	336
XXV SOME LESS COMMON FORMS OF SPECIFIC INFECTIOUS ARTHRITIS	342
XXVI NOTES ON SOME FORMS OF NONINFECTIOUS SPECIFIC ARTHRITIS	351

Part VI

XXVII NOTES ON SOME MISCELLANEOUS RHEUMATIC CONDITIONS	361
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Part VII—Pain in the Shoulder and Arm

XXVIII SUBACROMIAL BURSITIS	378
XXIX PERIARTHRITIS OF THE SHOULDER	388
XXX CERVICAL RIB AND THE SCALENUS ANTICUS SYNDROME	394
XXXI NERVE INVOLVEMENT AS A CAUSE OF PAIN IN THE CERVICAL SPINE SHOULDER, AND ARM	399

Part VIII—Low Back and Sciatic Pain

XXXII LOW BACK AND SCIATIC PAIN SOME GENERAL CONSIDERATIONS	411
XXXIII CERTAIN ANATOMIC AND PHYSIOLOGIC RELATIONSHIPS BEARING ON THE PATHOGENESIS OF LOW BACK AND SCIATIC PAIN	423
XXXIV SOME MEASURES OF TREATMENT FOR LOW BACK AND SCIATIC PAIN	432
XXXV THE CAUSES OF LOW BACK AND SCIATIC PAIN CLASSIFICATION	446
XXXVI THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN I MUSCULAR AND LIGAMENTOUS STRAINS AND SPRAINS	447
XXXVII THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN II POSTURAL STRAIN	453

XXXVIII. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: III. ARTHRITIS. IV. FIBROSITIS (MYOFASCITIS)	457
XXXIX. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: V. CONGENITAL ANOMALIES	463
XL. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: VI. ABNORMALITIES AT THE SACRO-ILIAC JOINTS	470
XLI. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: VII. AFFECTIONS OF THE PYRIFORMIS MUSCLE	479
XLII. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: VIII. CONTRACTURE OF THE ILIOTIBIAL BAND	481
XLIII. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: IX. ABNORMALITIES AT THE VERTEBRAL ARTICULAR FACETS	485
XLIV. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: X. PROTRUSION OF LUMBAR INTERVERTEBRAL DISKS	495
XLV. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: XI. THICKENED LIGAMENTA FLAVA	515
XLVI. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: XII. TUMORS—"RHEUMATISM REQUIRING MORPHINE"	519
XLVII. THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN: XIII. MISCELLANEOUS	522
GENERAL BIBLIOGRAPHY	527
INDEX	533

INTRODUCTION



INTRODUCTION

CHRONIC RHEUMATIC DISEASE ITS SOCIO-ECONOMIC ASPECT

If it be true that human misery loves company—it is in moderation, we hope. For the patient with rheumatism there is far too much company. In Massachusetts alone, in a survey of chronic diseases in that state in 1933, Bigelow and Lombard reported that 138,000 people (over 3 per cent of the population) suffer from chronic rheumatic conditions. An actual numerical count reveals more cases of chronic rheumatic disease than of Bright's disease, tuberculosis, and cancer combined. Chronic rheumatism, in Massachusetts, is eight times more prevalent than tuberculosis, twelve times more prevalent than cancer.

This situation is not peculiar to Massachusetts, however. The National Health Survey, conducted by the United States Public Health Service in 1935-36, indicated that of the 127 million persons in the country, 6,850,000, or approximately 5 per cent of the population, suffer from some form of rheumatism. The disease is indicated as being nearly twice as prevalent as its nearest rival, Bright's disease (3,700,000 cases), more than seven times as prevalent as "cancer and other tumors" (930,000 cases), ten times as prevalent as diabetes (660,000 cases).

It is not only the occurrence of the disease which is so alarming, but also its disabling effect, for, though it ranks first in incidence, it is second—not far behind—in producing disability and invalidism (permanent disability). That it ranks low (fourteenth) as a cause of death is feeble consolation. Rheumatism does not shorten life, it does, however, drain off much that lends value to it—buoyancy, health, and usually economic and financial independence, for which it substitutes discouragement, physical suffering, and dependence.

Again referring to the survey of the United States Public Health Service, rheumatism is our most costly national health problem aside from "mental and nervous diseases." We have in the United States a decrepit army of 6,850,000 rheumatic patients, whose care probably costs, directly and indirectly, infinitely more than did our national defense. For example, rheumatism was responsible for a loss from work of 97,200,000 days—more than any other chronic disease except the nervous and mental disorders men

tioned (which caused a loss of 132 500 000 days of work), and it produced more than twice as much disability as tuberculosis (which caused a loss of 41 400 000 days)

The invalidism (total disability) for which rheumatism is responsible is even more costly. As a cause of invalidism it again ranks second to only the nervous and mental diseases. The 147,500 persons permanently disabled by rheumatism in the United States represent twice the number of invalids (77 900) produced by all types of tuberculosis. The Massachusetts survey supported this conclusion, it indicated that 5,600 of the rheumatic patients in that state were totally disabled.

We have scant statistics of the actual cost in dollars and cents. The Metropolitan Life Insurance Company estimated the actual loss in wages resulting from rheumatism in one year at nearly \$250 000 000. The United States Veterans Bureau reported more than \$10 000 000 paid in pensions during 1936 to veterans suffering with chronic joint disease. The Chief Medical Officer of Health for England has reported officially that one sixth of the total invalidism of insured persons is attributable to rheumatic diseases and its economic cost to the country is not less than \$100 000 000 annually. These are considered to be conservative estimates. We have not taken stock of our increased burden in taxation, in higher insurance premiums and in the other ways in which most of us share the cost of maintaining these persons invalidated by rheumatism. It is a stupendous bill, yet, oddly enough, we meet it with relative indifference.

The statistics quoted refer to rheumatism in its broadest sense, by including the various types of "arthritis, gout, neuralgia, neuritis, lumbago, and so forth. From the standpoint of treatment, it is important to differentiate within this large, variegated group of rheumatic conditions, from the standpoint of invalidism and of the economic cost to each and every one of us individually, such segregation is irrelevant. To the public at large the significance of the problem lies in the fact that 5 per cent of the population of the United States is afflicted with some form of rheumatism.

More poignant is the fact that, as indicated by the United States Public Health survey, of all persons reported to have chronic joint disease, over 50 per cent were under forty five years of age, 70 per cent were under fifty five; and only 15 per cent were over sixty five years of age. Half of those afflicted with rheumatic disease are in the so-called productive years, a relatively small number are within the period of old age. Impairment of earning capacity therefore adds an economic burden to the physical encumbrance and suffering which the individual with arthritis is bearing. The sum total of the ravages of this insidious disease upon men and women in their prime of life is pathetic.

This fact is clear enough to anyone who sees many patients with chronic

arthritis, for the purely medical problem becomes a concomitant of the economic. The loss of time and earning capacity creates the dilemma of how to meet the economic burden of the medical care. It must be emphasized that for the person suffering from arthritis this economic burden results chiefly from such factors as loss of earning power from inability to continue to work, the frequent necessity for hospital and nursing care, the cost of professional physiotherapy, medication, and the like. In comparison with the cost of these features of the medical care, that of the physician's services are negligible. And so we have, coupled to the debilitating effect of the disease, the additional stress caused by the question of how to meet the economic problems created by the rheumatic disability. In many instances we find that this latter issue looms so large that its solution is essential before we can launch successfully on a course of treatment.

Unfortunately this situation is all too common, for, as has been shown by the recent National Health Survey, and as is well known from experience the burden of chronic disease falls most heavily on that part of the population which is least able to bear its full cost. Disability from chronic illness expressed in terms of the average annual number of days lost from work per person, is almost three times as great among families on relief, and twice as great among non relief families with incomes under \$1,000 as among families with incomes of \$3,000 or over.

Considering the magnitude of our problem, our relative apathy toward it is especially discouraging. It is only within the past twenty years, and particularly during the last decade, that the interest of the medical profession has been sufficiently aroused by the problem of rheumatism, so that some attention has been directed to it. There has been marked progress within these twenty years. We have not stumbled on any royal road to cure of these rheumatic disorders. We are arriving, however, at an integration of our knowledge of what can be accomplished in the treatment of these conditions, and even, to an extent, in their prevention.

To be sure, medical progress and discovery concerning the successful management of chronic rheumatic disease are today really far in advance of progress made in providing physical security for these patients with arthritis, yet interest in the problem of rheumatism is still far too limited. We cannot gloss over the fact that many medical practitioners are not yet fully cognizant of, and sufficiently oriented in the treatment of the patient with rheumatism. Too many of us approach the problem with indifference, scepticism, or discouragement. Comparing the facilities for the treatment of tuberculosis and rheumatism, Kling pointed out that the majority of rheumatic patients are under the care of a physician who shows little interest in this ailment, whereas the larger sanatoria and hospital departments are provided with the best facilities for the treatment of tuber

culosis and they are under the management of men who devote themselves to a study of this disease. If provision for satisfactory medical care of arthritis is not adequate if it is not readily available to all, it is inevitable that the patient in poor or even in moderate circumstances should be drawn to any source promising quick and inexpensive relief. The result of inadequate care may be a patient physically crippled, spiritually broken, and totally dependent economically.

To begin medical treatment anew at that time is to undertake it with a patient burdened not only by serious disease, but by impedimenta for which we ourselves are to an extent responsible. If we accept as corollaries the fact that rheumatic disease is most prone to occur among the less well to do that successful treatment of arthritis may require a long time, that there is not only a loss of earning power but the necessity for at least a preliminary period of hospital care then we must accept the axiom that the control of rheumatism is a problem not for the physician alone but one in which he must be aided by the public at large.

From the standpoint of the patient or of society nothing short of complete eradication of the disease when possible can be thoroughly satisfactory. Too often there is a recurrence of the disease when the patient resumes work too early, that is before the disease is completely stamped out. Such recurrences may turn out even more disastrously than the initial attack, they may require even longer periods of hospitalization and treatment, and consequently may prove to be a greater economic burden on the patients and on society. The investment that society would need to make for the provision of adequate medical care in arthritis may seem large, but it would be more than amply repaid. Unfortunately we have never stopped to calculate the present staggering cost of the disease. If we had, our social conscience would have speedily remedied our oversight.

Recognizing the need for concerted action in the prevention and cure of rheumatic disease, organizations, composed of physicians interested in the problem, have been created in various parts of the world. They are eager to organize the necessary facilities for adequate solution of the problem. The activities of the British Committee for the Control of Rheumatism and of the American Rheumatism Association have already proved fruitful. Their work, however, has just begun. Progress based upon the efforts of these physicians alone cannot be rapid, nor altogether successful. They must have not only the assistance of the medical profession at large but in addition, the aid of the public. We must establish a broad program for educating people concerning the nature of rheumatic disease, concerning effective methods for its prevention and control, and concerning the effectiveness of medical treatment as we know it today.

The survey by the Massachusetts Department of Health indicated that

of the people subject to various chronic illnesses, including rheumatism, only 31 per cent were under the care of physicians, the rest were either receiving no treatment whatever, or were treating themselves. Not realizing how disabling chronic rheumatic disease can be, if neglected, some of these patients thought their condition not serious enough to require medical attention. Others, perhaps, had been through one or more desultory courses of treatment without noticeable improvement, they felt a physician could not help them. There is reason to suppose that many of these patients might have improved had they received the full benefit of all that is known today concerning the management of chronic arthritis.

The family physician should realize that he occupies a pivotal position of responsibility toward the arthritic patient. His attitude toward the problem of arthritis and his accomplishments in this field will determine to a large extent whether the movement for the control of the rheumatic diseases will be successful or whether the stream of unhappy arthritics, made more wretched by desultory, slipshod treatment, will flow toward the cultists.

How, concretely, can a successful program for the control of rheumatism be carried out? It is pretty much agreed that in most cases of arthritis (especially in active cases of atrophic arthritis) the best place to initiate treatment is in the hospital. A period of hospitalization not only permits the application of certain therapeutic measures not readily employed in the home or in the physician's office, but affords opportunity for educating the patient with regard to measures that should be carried out at home later on.

But as Kling pointed out, hospital facilities for the care of rheumatic patients are at present woefully inadequate. He deplored the fact that everywhere he encountered the greatest difficulty in securing even urgently needed hospitalization for patients afflicted with rheumatic disease and arthritis. Analyzing statistics obtained from a large number of hospitals in various parts of the country, he found that the number of admissions for arthritis and rheumatic diseases, in the hospitals surveyed, represented only about 1.3 per cent of the total admissions. The number of patients with rheumatic diseases admitted to hospitals in the United States is probably less than 10 per cent of those totally disabled by arthritis. Moreover, this hospital survey included a number of institutions having departments carrying on research in rheumatic diseases and therefore admitting more than the average proportion of rheumatic cases. On the whole, the general hospitals are not inclined to accept rheumatic patients. Their facilities are often taxed by urgent surgical cases or those with more acute medical problems, such cases are given preference because they require shorter periods of hospitalization and, therefore, permit a greater turnover.

This, however, is not the only reason. A disease like tuberculosis also

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requires a long period of hospitalization. Nevertheless, there are in this country about 87 000 beds set aside in special institutions and in departments of general hospitals for the treatment of tuberculosis. The reason is obvious. Tuberculosis is communicable and the general public has been educated to an awareness of the danger. True arthritis is not communicable as a disease but the burdens imposed by it, including the cost, are communicated to and shared by a large portion of the population.

It is no doubt partly because interest in the problem has not been sufficiently aroused that there is only one institution in this country which has a group of beds set aside especially for the treatment of arthritis. Kling justifiably deplored the fact that of his own clinic patients hardly 1 out of 200 or one half of one per cent, were able to secure free admission to a hospital for treatment. The large proportion of patients with arthritis who are admitted to hospitals are private patients. In contrast, about 90 per cent of the beds for tuberculous patients are free and only 10 per cent paid. This is a proportion more in keeping with the requirements of the situation.

Davidson and Duthie indicated the existence of a similar situation in the British Isles. Most patients with arthritis they pointed out, are sent to the hospital only when they have become markedly incapacitated. These authors regretted the failure on the part of the medical profession and society to recognize the fundamental need for institutional treatment in the early stages of the disease when deformity can be prevented and complete cure in many cases achieved. Of the last 100 cases of rheumatoid arthritis admitted to the wards of the Aberdeen Royal Infirmary, Davidson and Duthie pointed out, the average duration of symptoms before admission was three and one half years. It is not surprising they add, that in a large proportion of these cases cure is impossible and a limited restoration of function is all that can be attained. Hence the outlay of time and expense on these late cases cannot be justified on economic, but only on humanitarian grounds. They voice the general agreement of all those conversant with the situation when they say that 'not until sufficient beds are available for the treatment of cases in the early stages and for adequate periods will the present gloomy outlook toward the treatment of the chronic rheumatic diseases be altered'.

These authors, too, hold the present attitude of the general hospital toward rheumatic disease accountable for a large share of the ignorance and apathy of the medical profession in regard to the national importance of the problem of rheumatism and the insufficient supply of graduates interested in and suitably trained for the treatment of the chronic rheumatic diseases. Such criticism is also applicable to the general hospitals in the United States. The creation of special departments for the care of rheumatic patients has lagged far behind the evident need for them.

The problem is obvious and so are the requirements for its solution. If adequate steps to control the havoc wrought by crippling rheumatic disease are to be taken special institutions and departments in general hospitals will have to be created for the care of these patients. It will be necessary to equip these departments with the facilities required for the successful management of arthritis. At present with these facilities rather loosely and inefficiently organized the cost of providing them is relatively high. They could be provided at a relatively low cost in special institutions or departments.

Criticism might be advanced against the practicability of hospital care of patients suffering from rheumatism. It may be argued that the extensive length of time generally required for the cure of rheumatoid arthritis precludes the possibility of extending hospital facilities to all patients who may require them. But this criticism does not necessarily hold. Although it is true that the course of treatment of an arthritic patient from beginning to end to the time of rehabilitation may require many months or even a year or two we have not found it necessary to hospitalize these patients for the full duration of their illness.

A practical way of handling the situation and one that has proved very satisfactory in our experience is as follows. The patient is hospitalized for a preliminary period of several weeks or months depending upon the complexity or severity of the problem presented. During this time the foundation for the entire course of treatment is laid. In addition to providing the patient with adequate rest we attempt to establish his emotional adjustment to the requirements imposed by the disease. The attempt has often proved amazingly successful. We have known patients to undergo a veritable transformation. With the dissolution of fears and anxiety they gain confidence in the whole program of treatment and develop determination to aid in the process of recovery. At the same time the necessary orthopedic measures are provided for the prevention of deformity or if it exists for its correction. Surgical procedures requiring hospital facilities are carried out. These include blood transfusions, removal of infective foci when indicated and other necessary operative treatment. Physiotherapeutic measures are instituted and either a relative of the patient or a nurse is instructed in the particular physiotherapeutic measures required. This is accomplished by having such persons accompany the patient to the physiotherapy department where they are trained as safe amateur physiotherapists and are prepared for the job that is ahead when the patient returns home.

When he has made a start toward recovery the patient is returned to his home. During the preliminary stay at the hospital he has learned that the period of hospitalization is not expected to effect a cure but to serve as a running start toward recovery and that treatment must be continued at

home with perseverance and accuracy until the fullest degree of recovery possible has been attained. When measures for the prevention or correction of deformities must be continued at home, provision is made for the patient to purchase or rent the necessary physical equipment, including the proper type of bed, traction apparatus, weights, and so on.

It is to be hoped that eventually such advantages will be open to all arthritics. Since hospital facilities are still relatively limited, selection of patients for admission could be made largely on the basis of the prognosis for complete cure of the disease and rehabilitation of the individual. Such criteria for the selection of patients for hospital care would offer an inducement to seek medical treatment early. Other cases could be selected, in addition, for research or teaching purposes.

Of course physical facilities alone are not enough. Without appropriately trained personnel such a program would be worthless. The number of physicians interested in or adequately trained in the field of rheumatology is altogether too small and inadequate to meet present day needs, for not only is the number of patients who require expert care large, but increasing effectiveness of therapy makes a much wider application of these measures necessary. We need physicians who have in addition to an interest in rheumatic disease the training required for its diagnosis and expert treatment. Davidson and Duthie have found that many physicians on the staffs of the voluntary hospitals have little interest in the group of rheumatic diseases and not infrequently dislike having such cases under their charge. This attitude is explained partly by inadequate knowledge of modern methods and the good results which may follow these procedures and partly by the difficulty of providing bed accommodation for periods sufficiently long to achieve optimal improvement.

Rheumatology is undoubtedly one of the most difficult of the medical specialties and proficiency in this field is derived from special training and disciplined experience. Training in the principles involved in the diagnosis and management of the rheumatic diseases requires first a view of medicine as a whole from a broad perspective, for no disease, except perhaps syphilis and tuberculosis, touches more upon every phase of general medicine than do the rheumatic conditions. In addition to thorough grounding in general medicine, there is required, of course, an especially wide and practical experience with the principles and methods employed in the examination and treatment of arthritis. These include, among other things, a practical knowledge of accepted physiotherapeutic principles and methods, and a close familiarity with the principles of operative and non-operative orthopedics applicable to the treatment of arthritic joints. Only through such knowledge is the physician able to co-operate effectively with the orthopedist who may contribute materially to the well being of the patient.

with arthritis Obviously, training to acquire proficiency in all of these branches can be achieved best only by long time residency in hospitals with an interest in the care of arthritis

The institutions described would have a salutary effect beyond that which accrues directly to the rheumatic patients hospitalized, for such institutions would also constitute centers for further research in the rheumatic diseases They would also provide the necessary centers for the training of a number of physicians who would take their place in extending the benefits of present day rheumatology to a much larger number of those afflicted than is now possible

With the organization of arthritis departments in general hospitals or special institutions, it should be possible to establish teams of "wandering" physiotherapists who could make frequent rounds among the patients, bedridden at home, to administer treatment at nominal charges In this way professional physiotherapy could be made available to those patients who need it for long periods after dismissal from the hospital When necessary, trained assistants, attached to the staff of the arthritis department, could also make periodic home visits to such patients in order to provide the necessary medical care Or they could cooperate closely with the patient's attending physician and help in guiding the course of treatment

Finally, the arthritis department of the hospital could also serve as a post graduate teaching center for the general practitioner of medicine, who is the first line of defense against the ravages of arthritis Just as in the control of tuberculosis, so also in the control of the rheumatic diseases, the family doctor is in the forefront of the attack The great prevalence of the rheumatic diseases, and the natural tendency for the patient with early manifestations to consult his family doctor, force the general practitioner into the most strategic position with reference to the early control of these conditions The family doctor must be prepared for this task, for the decisive battle in the control of arthritis will be waged by him If he is to succeed, recent advances in knowledge of the disease and the newer methods of treatment must be made available to him

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PART I

ATROPHIC ARTHRITIS

CLASSIFICATION OF THE ARTHRITIDES

ATROPHIC ARTHRITIS SOME BASIC CONSIDERATIONS

THE PRECIPITATING ETIOLOGIC FACTOR IN ATROPHIC ARTHRITIS

PATHOLOGY OF ATROPHIC ARTHRITIS

PATHOGENESIS OF ATROPHIC ARTHRITIS

FOCAL INFECTION IN ATROPHIC ARTHRITIS

VARIOUS FOCI OF INFECTION IN ARTHRITIS

ATROPHIC ARTHRITIS CLINICAL MANIFESTATIONS AND DIAGNOSIS

ATROPHIC ARTHRITIS OF THE SPINE

STILL'S DISEASE

PSORIATIC ARTHRITIS

FOCAL ARTHRITIS

TREATMENT OF ATROPHIC ARTHRITIS

CHAPTER I

CLASSIFICATION OF THE ARTHRITIDES

If we are to treat any group of conditions rationally not merely symptomatically our first concern in approaching any given case is to classify it as accurately as we can into the category in which it belongs. When possible the classification should be on an etiologic basis for that furnishes the most direct guide to treatment. Arthritis is no exception to this rule. At present however the etiology of all types of arthritis is not definitely known. For the time being therefore we must adopt a presumptive etiologic classification for those types in which a specific one cannot be employed.

The classification of arthritis requires clarification because it has in the past been so burdened with a variety of nomenclatures. The profusion and looseness of the terms employed in the classification of joint diseases have been actually bewildering to the physician. Mere recognition of that fact however does not eliminate the difficulty. For obviously one cannot discuss arthritic disease without understanding the precise meaning of the various designations so often employed.

The American Rheumatism Association has urged adoption of a simple standard nomenclature. Until that suggestion is universally accepted the physician must be familiar with at least the more common of the numerous terms he now encounters in the literature redundant and inaccurate as some of those terms may be. Not only is there need for familiarity with the terminology employed but also with the criteria—clinical and pathologic—upon which the chosen terminology is based. This is particularly applicable in a consideration of the two largest groups of chronic rheumatic disease which in conformity with the suggestion of the American Rheumatism Association we designate *atrophic* and *hypertrophic* arthritis.

For each of these two common types of chronic arthritis at least six synonyms frequently appear in the literature. When we enter upon discussion of the various types of rheumatic disease we will indicate the basis for the designations we employ and of the synonyms most likely to be encountered.

And now we will outline a deliberately broad and simple classification of arthritic disease. It should prove helpful to the physician who has in the past been only confused by too many cumbersome details.

CHAPTER I

CLASSIFICATION OF THE ARTHRITIDES

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I JOINT DISEASES THE ETIOLOGY OF WHICH IS NOT SPECIFICALLY KNOWN

ATROPHIC ARTHRITIS

Synonyms

{	Rheumatoid arthritis
	Infectious arthritis
	Proliferative arthritis
	Primary progressive polyarthritis
	Chronic rheumatic arthritis
{	Arthritis deformans

Includes the following forms of manifestations

- Typical atrophic arthritis
- Atrophic arthritis of the spine
- Still's disease (in children)
- Psoriatic arthritis
- Focal arthritis

HYPERTROPHIC ARTHRITIS

Synonyms

{	Osteoarthritis
	Degenerative arthritis
	Senescent arthritis
	Climacteric or menopausal arthritis (in women)
	Osteoarthrosis
{	Arthritis deformans

Includes the following forms of manifestations

- Diffuse hypertrophic arthritis
- Heberden's nodes
- Malum coxae senilis (hypertrophic arthritis of the hip)
- Hypertrophic arthritis of the spine
- Static arthritis
- Menopausal arthritis

MIXED FORMS OF ARTHRITIS (page 371)

RHEUMATIC FEVER (page -91)

II JOINT DISEASES OF SPECIFIC ETIOLOGY

METABOLIC GOUTY ARTHRITIS

INFECTIOUS

- Gonococcal arthritis
- Tuberculous arthritis
- Acute suppurative arthritis
- Pneumococcal arthritis

Meningococcal arthritis
 Arthritis of scarlet fever
 Syphilitic arthritis
 Arthritis of Brucellosis
 Arthritis of Haverhill fever
 Arthritis associated with lymphogranuloma venereum
 Arthritis associated with ulcerative colitis
 Arthritis of typhoid fever
 Tuberculous rheumatism
 Other forms of specific infectious arthritis

TRAUMATIC ARTHRITIS

CONSTITUTIONAL HEMORRHILIC ARTHRITIS

ALLERGIC ARTHRITIS OF SEVERE SICKNESS

NEUROPATHIC JOINT DISEASE

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[For a list of books and monographs dealing with the general aspects of
 chronic rheumatic disease (including considerations on the subject of the
 present chapter) see page 527]

CHAPTER II

ATROPHIC ARTHRITIS

SYNONYMS *Rheumatoid arthritis infectious arthritis proliferative arthritis primary progressive arthritis chronic rheumatic arthritis arthritis deformans*

SOME BASIC CONSIDERATIONS

Chronic arthritis is now better understood than ever before. Gaps of which the student of arthritis is consciously aware still exist in our knowledge but the practitioner today can approach the handling of the arthritic patient with confidence in therapeutic resources based on more than hazy hypotheses. The knowledge we have already gained has given us not only a rational approach to the study of the patient with arthritis but to a useful system of therapy. Properly applied it can accomplish for these unfortunate patients as much as if not more than can be achieved by appropriate therapy in any other serious chronic disease.

NOMENCLATURE

Atrophic arthritis is the name accepted by the American Rheumatism Association for the type of joint disease under discussion. It is one of the most common and most serious forms of rheumatic disease. The British and many American writers prefer the designation rheumatoid arthritis for the same condition. Many of those who believe the condition to be precipitated by infection refer to it as infectious arthritis. Still others speak of it as proliferative arthritis referring to the earliest pathologic evidence of cellular infiltration and proliferation of synovial membrane into a pannus. The term is not employed in the same sense as hypertrophic arthritis which refers to the earliest bony exostoses in degenerative arthritis. Perhaps it may seem paradoxical to speak of the same type of arthritis as proliferative and atrophic but the distinction is clear if it is recognized that the former adjective is applied to descriptions of the synovial and periarthritic changes in atrophic arthritis and the latter to those of the earliest cartilage and bony changes for although there is proliferation of

the synovial tissue, the earliest change in the cartilage is destruction of it, and in the bone, atrophy from decalcification

"Primary progressive polyarthritis" is a term that, fortunately, is passing out of use. It has nothing to recommend its survival. It is basically incorrect for it presupposes an inevitable tendency to progression of the disease which, particularly if proper treatment is employed, need not occur.

"Chronic rheumatic arthritis" is another term seldom seen nowadays. It is altogether too meaningless ever to be employed, as also is the term "arthritis deformans," since many types of joint disease, if untreated, may lead to deformity. As a matter of fact, the term "arthritis deformans" has been confusing, being employed by some writers as synonymous with "atrophic arthritis" and by others as synonymous with an entirely different form, "hypertrophic arthritis."

DEFINITION

The characteristics of atrophic arthritis which distinguish it from other types of chronic joint disease will become clearer as we discuss its pathological and clinical manifestations. For purposes of orientation, however, we indicate here, in a most general way, what we shall include within the group "atrophic arthritis."

Briefly, atrophic arthritis is the type of joint disease of acute or insidious onset, affecting chiefly young adults. There is a tendency to involvement of multiple joints, frequently symmetrically. If the hands are affected, there is a predilection for the metacarpophalangeal and middle phalangeal joints, the latter producing the spindle shaped fingers. There is a tendency to early deformity and, later, ankylosis, unless steps are taken to prevent them.

Probably the most distinctive clinical feature of atrophic arthritis, as far as the joints are concerned, is the early periarticular involvement. In advanced cases, when much damage has occurred, the joints may reveal extensive destruction of the cartilage and bone, and even secondary hypertrophic bony changes adjacent to areas of bone destruction. However, at this stage the swelling and thickening of the periarticular structures are likely to be even more pronounced, revealing the basic nature of the disease. Evidence of constitutional debility, strikingly suggestive of infection, appears to some extent in practically all cases.

Atypical offshoots of the more usual pattern, but those which we shall nevertheless consider in the general group of atrophic arthritis, are cases with monoarticular affections or with involvement of two or three large joints and with relatively little constitutional debility. This group is segregated by some as a distinct clinical entity, which they call "focal arthritis." We shall consider the form of arthritis occurring in children (Still's disease) and

the ankylosing type of spondylitis as two additional variants of the more common pattern of atrophic arthritis

Atrophic arthritis is a disease which extends far beyond the confines of the articular structures, to the patient as a whole, leading to many and varied manifestations. In atrophic arthritis the complete organism is ill. The involvement of the joints represents merely an extension of the disease to only one group of organic appendages. It is an important group, to be sure, from the standpoint of their usefulness to the individual. Because of the havoc to the patient's life that may be wrought by the joint involvement, the consideration of rheumatic disease was, for centuries, focused sharply on the articulations alone, with little or no regard for what was happening behind the face of the clock. In recent years closer study has revealed the fuller significance of many phases of this disease, totally neglected in the past.

In the last two decades more progress has been made in the understanding and treatment of rheumatic disease—particularly of atrophic arthritis—than had been made in all preceding years. True, there have been no spectacular discoveries, such, let us say, as the discovery of the clinical virtues of sulfanilamide. The progress that has been made in the treatment of atrophic arthritis was not based upon any single revolutionary discovery, either of its cause or of a specific cure, but arose from a sound evaluation of the meaning of the many facts that we have come to know concerning this disease. The integration of the many isolated observations is not a simple matter. An imaginative interpretation of interrelationships is still required to fill in the gaps, imagination, it must be emphasized, rigidly disciplined. Under such conditions it is possible even today to evolve a general concept of chronic arthritis that will be accurate and enduring. No doubt the future will sharpen certain phases of the concept, and translate our generalities into specific details. For that we must wait.

Although our ultimate aim is to devote most of our attention to the matter of therapy, that phase of the subject can hardly be discussed intelligently without at least a sketchy review of what the concept "atrophic arthritis" embraces. We must, of necessity, stress only the highlights of the subject.

THE CONSTITUTIONAL BACKGROUND FOR ATROPHIC ARTHRITIS

Because involvement of the joints is the most striking effect of arthritis, many fundamental aspects of the disease have gone unrecognized. Its constitutional foundation—a most important factor—is not generally con-

sidered, yet it can be seen readily enough by any physician who will probe for more than superficial manifestations

One may well ask Is the arthritic destined for his disease? The idea of constitutional susceptibility to disease is no longer new Through the centuries clinicians had repeatedly been impressed by something of the individual's "diathesis" in its relation to his disease We have long been familiar with such terms as 'phthisic habitus,' and 'fair, fat and forty,' which hint at the relationship of physical structure to disease Nevertheless, the factor of constitutional predisposition is generally given so little consideration in medical practice that I feel it justifiable to digress just enough to point out some recent investigation bearing on this topic

The influence of human constitution in disease is readily apparent through analysis of the fate of *monozygotic* twins The many instances which have been reported of the occurrence of identical disease in identical twins, stress the importance of the soil in relation to the development of a specific disease Take the striking example of the twin sisters reported by Wolfsohn One lived in San Francisco, the other in New York, both simultaneously developed diabetes in their fifty second year and both died of cerebral hemorrhage within a short time of each other The brothers reported by Trousseau were aware of a similar inherent tendency "I am now having my rheumatic ophthalmia," one wrote his twin in a far distant city, "you must be having yours" The inherent constitutional basis for disease is best exemplified, perhaps, in the case of twins reported by Kretschmer Within seven months of each other, both developed renal tuberculosis, preponderantly in the right kidney where the inherent weakness evidently lay, disease did not result until this weakened structure in each of the twins met with the invading organism, the lungs, and other sites equally open to the attack of the organism, were, however, significantly capable of escape

By detailed anthropometric and psychologic investigations on clinical material comprising a variety of disease entities, Draper has in recent years been able to correlate, to an extent, certain distinct constitutional potentialities with certain definite diseases The work of Petersen and Levinson enlarged upon the possible indices of human potentiality, by 'seeking to define constitution in terms of measurable biologic reactions, rather than in developmental attributes' Still other means of appraising various types of human constitution must exist, for only a few threads in the material have been discovered Stimulated by these studies, those of the future, it is hoped, will trace all the strands in that intricate pattern, the individual make up When this has been achieved it is likely that adequate clinical investigation of a patient's disease will be preceded by analysis of the individual himself

The foregoing considerations offer one logical explanation for the varia

tion in the natural course of arthritis in different patients. Some with an initially severe acute attack will eventually be entirely well without a trace of the previous joint involvement; others fortunately a much smaller number develop in a short time irreparable damage to joints with ankylosis despite everything that may be done. This situation is not unique in arthritis. It is not unlike the difference between slowly progressive pulmonary tuberculosis and fulminating caseous pneumonia that may be so quickly fatal. It must be then that behind the scenes of chronic disease there is a determining mechanism of varying susceptibility. In arthritis as in any other disease we must examine the background of the process. Any consideration of arthritis is incomplete if on viewing the many extrinsic factors that are undoubtedly concerned one fails to recognize the underlying constitutional factors in the patient for the entire course and the eventual outcome of the disease may be predicated to a large extent upon the patient's susceptibility as well as upon other extrinsic factors.

Concretely constitutional vulnerability to arthritis probably depends on such factors as inherently inferior joint tissues, impaired blood supply, the type of nervous system with which the potentially arthritic subject is endowed, perhaps abnormalities in body build and in the form and function of the gastrointestinal tract and increased susceptibility to certain infections. These and probably many other such influences determine in a large measure whether arthritis will occur at all and modify the course of the disease.

Superimposed upon this groundwork are many extraneous influences which again modify both the degree and the kind of arthritis that develops. Thus the foundation upon which arthritic disease develops and thrives is further shaken by a number of factors such as fatigue, exposure to damp and cold and nutritional disturbances, particularly those associated with vitamin deficiency and dysfunction of the gastro-intestinal tract. The soil so to speak is therefore only too well prepared for the seed of the disease which probably originates from some focus of infection. It might undoubtedly be the inherent tendency to the disease plus the influence of the factors mentioned which mark the patient with atrophic arthritis for infections which others harboring these identical potential sources escape.

We do not yet know all the factors inherent in the potentially arthritic patient. Arthritis is not strictly speaking an hereditary disease yet pre-disposition to it seems to occur in family groups. Pemberton found direct or collateral hereditary tendencies to arthritis in 58 per cent of a series of cases he observed. Those seemingly susceptible may present a rather uniform type of body configuration, some of the characteristics of which are long slender bodies, a tendency to visceroptosis, cold, sweaty hands and a high strung emotional disposition (Fig. 1). It must be understood however that

persons of this type are not inevitably destined for this disease, nor are others of different body conformation and emotional disposition always immune. This apparent vulnerability to atrophic arthritis among several

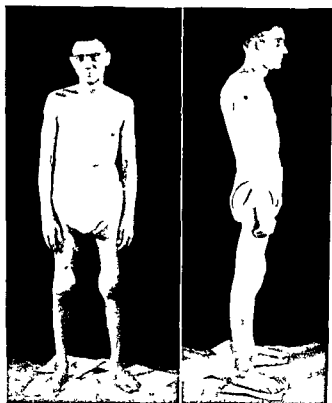


FIG. 1 The typical candidate for atrophic arthritis. Note the asthenic habitus and poor posture. Note also the spindle-shaped legs from persistent hydrops of the knees and atrophy of the muscles of the legs.

members of a family, however, particularly among those who show some uniformity in physical and nervous constitution, suggests that some constitutional element inherent in the individual constitutes a base upon which arthritic disease develops.

If it is accepted that, to a certain extent, the general groundwork for the arthritic syndrome is inborn and irrevocable, it must follow that in the treatment of such subjects we must aim not only to mitigate the effects of extraneous deleterious influences, but also to shelter the patient by creating for him a protective environment in which his innate susceptibilities toward arthritis will be least exposed to attack. And so the adequate treatment of arthritis implies more than the mere use of analgesia and physiotherapy, and a supposedly specific vaccine. It must include a careful

scrutiny of the individual as a whole an appraisal of his constitutional background and finally an all embracing program of treatment aiming at every phase of the disease

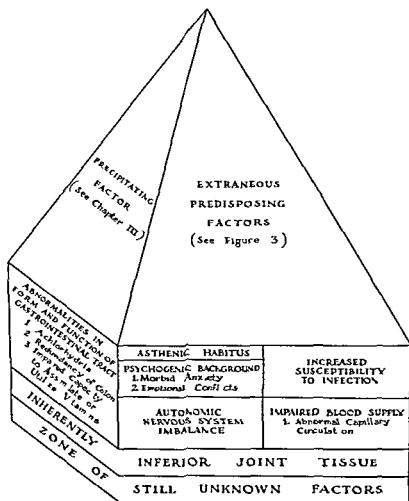


FIG. 2. Constitutional basis upon which atrophic arthritis is built. Without the base the pyramid does not exist.

SOME STIGMATA OF THE ARTHRITIC CONSTITUTION

What are some of these constitutional stigmata in arthritis? Unfortunately study of this aspect of the problem has not yet progressed far enough to yield the specific details and refinements that no doubt await our discovery in this, as in other diseases. The nearly universal occurrence of achlorhydria in pernicious anemia, although known for years was not recognized formerly, as it is now, as one very important constitutional

aspect of that condition. In the same way we probably miss recognition of many stigmata of the arthritic constitution which we might identify if we but looked for them. Certain features, however, are so striking that we cannot fail to recognize them even now (Fig. 2).

Asthenic Habitus

We have already mentioned the general asthenic habitus of the patient suffering from atrophic arthritis, with the tendency to visceroptosis, which is clearly one gross manifestation of the constitutionally poor make up of the potentially arthritic individual.

Autonomic Nervous System Imbalance

Another manifestation is the impressive evidence of autonomic nervous system imbalance in a large proportion of patients with atrophic arthritis. That this condition is not a result of the disease, but more likely a precursor of it, is evident in the existence of such a state of autonomic imbalance for years before the onset of the disease.

The concept of autonomic imbalance is still not sharply defined, but its expressions, in certain physiologic abnormalities, are glaringly apparent in the patient with arthritis. The cold, pale or mottled bluish, sweaty extremities are probably the result of an abnormal capillary circulation, mediated through disturbed function of the autonomic nervous system, and in the vast majority of such cases, this vasomotor and secretory abnormality has been in evidence, to some degree, for many years preceding the onset of the arthritis. In many of them it dates back to childhood.

That this constitutional defect is in some way related to the arthritic syndrome cannot be doubted, but just how it operates we cannot yet say. It is not likely that its effect is purely mechanical, leading simply to deficient oxygenation of tissues, it probably has other more fundamental physiologic effects and "tie ups" which deserve close study.

Other Physiologic Abnormalities

Pemberton has pointed out the decreased capacity for the utilization of glucose by the tissues of the patient with arthritis. This abnormality may not be a fundamental defect, but may perhaps be either an expression of some more deep seated physiologic aberration characteristic of the arthritic constitution or the effect of an abnormal capillary circulation.

The Digestive Tract and Vitamin Deficiency

In recent years study has been devoted to the condition of the colon in patients with arthritis. Since considerable variation in the size and function of the colon is observed in many seemingly normal individuals, the finding

of abnormalities in the large bowel in chronic arthritis was for a long time not regarded seriously. Experiments indicated however that animals in whom abnormalities in the large bowel have been induced by deprivation of vitamin B exhibit among other things a heightened tendency to arthritic and other bony changes. Suspicion was then aroused of a possible relationship between the form and function of the gastro-intestinal tract and atrophic arthritis in man.

Studying patients with arthritis particularly from the standpoint of the condition of the large bowel Fletcher observed roentgenographic evidence of striking dilatation and redundancy of the colon in many of them. Employing large rations of vitamin B in the treatment Fletcher noted clinical improvement which could be correlated with a return to a normal appearance of the colon. In some cases the dilatation of the bowel disappeared entirely and the previously redundant sigmoid became normal in outline. Haustration previously absent in some cases returned. Pemberton and Peirce were able to induce clinical improvement of the arthritis and in the condition of the colon by temporary elimination of all carbohydrate and by the gradual resumption of a diet extremely restricted in the quantity of carbohydrate.

Just what the significance of these striking changes in the colon may be is not yet clear. Whether they antedate the arthritis and are causally related to its development is unknown. It has been suggested that these changes in the large bowel are the result of vitamin deficiency, that the tendency to the development of vitamin deficiency may be increased by the consumption of large rations of carbohydrate. It may also well be that the vitamin deficiency and the abnormality in the colon are interrelated and in turn dependent upon a more fundamental abnormality of the arthritic constitution.

It is not to be assumed that only the lowermost portion of the digestive tract is singled out. The tendency to achlorhydria among individuals with arthritis is no less striking even though its genesis may be even less clear.

Psychogenic Makeup of the Candidate for Arthritis

It has been noted that in the psychogenic sphere also patients with arthritis present certain constitutional traits which are fairly distinctive for the group. The evidence here is only fragmentary; this aspect of human constitution has not yet been studied intensively enough in relation to arthritis or to physical disease in general and yet there are in the medical literature many allusions to the probable importance of this aspect of the arthritic patient's constitutional endowment.

Lilman and Mitchell in studying a group of patients with atrophic arthritis were impressed by the tendency toward morbid anxiety noted in

many of them either before or following the onset of the disease. Here is a psychogenic factor accorded little consideration by the practicing physician, though it enters unquestionably and in a very concrete way in facilitating the onset of the disease; it undoubtedly impedes recovery and increases the tendency to recurrence or relapse. Such evaluation of the patient's inherent psychologic makeup is emphatically not idle indulgence in nebulous or purely philosophic exercise. On the contrary, the physician is here indeed on firmly practical ground. We may wonder, for example, what the effect of such an abnormal anxiety state may be on an inherently poor sympathetic nervous system. We may surmise the havoc such a psychologic makeup may wreak on an organism inherently given to excessive fatigue and exhaustion, to gastrointestinal dysfunction and poor resistance to infection.

Although this cursory analysis of some aspects of the constitutional background of atrophic arthritis bristles with qualifications and uncertainties, it cannot be ignored without obscuring a complete view of the background of arthritic disease. It may also serve to suggest at least that along with such relatively obvious constitutional anomalies there may go other physiologic disturbances. These may be hidden far in the substratum of the arthritic constitution, but they may nevertheless play a determining part in predisposing the patient with arthritis to the infections and intoxications to which he falls prey, and likewise a similar mechanism may determine the varying degrees of vulnerability of the articular structures to infection and injury from various types of noxious agents.

SOME EXTRANEOUS PREDISPOSING INFLUENCES

If we assume that those elements of human constitution to which we have alluded determine the soil upon which arthritic disease develops, then we must examine certain conditions that prepare this soil for the actual seed of the disease (Fig. 3).

It is not difficult to perceive the adverse effect of a cold, damp climate on a patient with a constitutionally impaired vasomotor mechanism and poor capillary circulation. Such climatic conditions, to which the average individual can adjust himself by virtue of a normal vasomotor mechanism, upset the physiologic equilibrium of the potential arthritic whose vasomotor apparatus is defective. This may lead to lowering of the general resistance to infection and may lead also to abnormal physiologic conditions in joints and muscles, making them vulnerable to injury. The simulation of climatic conditions producing this effect may be the explanation for the higher incidence of atrophic arthritis among those living under poor housing conditions and among those who through occupation are continually exposed to cold and wet.

Because any physical hardship which may lower resistance is conducive to preparation of the potentially arthritic patient for his disease, we find atrophic arthritis most common among those exposed to overwork, worry,

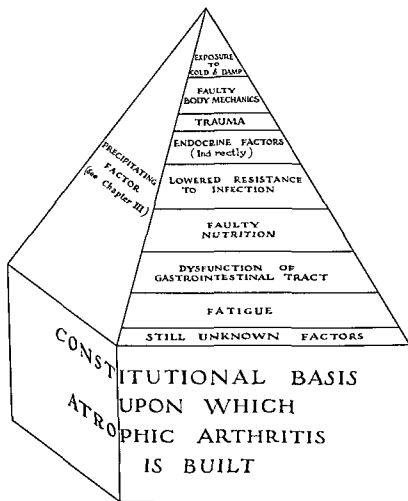


FIG 3 Extraneous predisposing influences in atrophic arthritis

anxiety, nervous shock, debilitating illnesses of various sorts, unhygienic surroundings, and economic handicaps. The latter may act through an effect on nutrition or by intensifying an inherent tendency to anxiety and to instability of the nervous system.

The predisposing factor most frequently met in atrophic arthritis is physical debility from any cause, particularly from overwork, from nervous shock, and the like, and more particularly if these be associated with prolonged or undue anxiety. The writer entertains no doubt about the direct

relationship between nervous shock and the onset of the disease, having so often seen a violent attack of widespread atrophic arthritis developing within a few days or a week or two after an explosive nervous shock precipitated by disastrous misfortune in the patient's family. With the least prodding, and frequently voluntarily the patient himself is likely to express a belief in the close relationship between such nervous shock and the onset of his arthritic disease.

A valuable objective analysis of the relationship between emotional stress and attacks of atrophic arthritis was contributed recently by Cobb, Whiting and Bauer. Studying fifty patients with atrophic arthritis they found impressive evidence of the fact that in no less than 66 per cent of them there was demonstrable chronological correlation between emotional stress and the attacks of arthritis. In their series unhappiness within the family was the environmental burden most frequently associated. Next in frequency were severe financial worry and the loss of a parent or spouse; these followed by other factors less easily classified. Such specific data leave little or no room for doubt that the emotional factor is a formidable one in preparing the soil for arthritic disease, and because it conditions the entire course of the disease the factor of emotional stress must be given adequate consideration as one of the important elements to be contended with in treatment.

Even carrying the body in the erect position seems too much for the constitutionally inadequate, asthenic body makeup of the candidate for arthritis. He becomes literally bowed under this physiologic stress, this results in further impairment of many physiologic functions, adds to inherent fatigue, and increases susceptibility to infection. Sooner or later most of these people develop static abnormalities in the feet which contribute not only to intensifying general fatigue but to microtrauma on weight bearing joints, which are thus left wide open for attack by toxic or infectious agents.

We have hinted at the possibility of inherited abnormalities in the form and function of the gastro intestinal tract: redundancy of the colon, achlorhydria, perhaps an impaired capacity to assimilate or utilize vitamins. Superimposed upon those possibly inherited traits, and perhaps resulting from them are various types of gastro intestinal dysfunction so often encountered by the patient before or after the onset of the disease. There may result, then, various grades of disturbance of nutrition such as are frequently associated with states of vitamin deficiency. The ramifications of these abnormal nutritional states may extend to other vital physiologic functions and thereby aggravate fatigue, susceptibility to infection, and so on. How intertwined and interdependent are the innate and environmental stresses which may conspire to produce the potential arthritic candidate is obvious.

In the mention of microtrauma on weight bearing joints we have inti-

mated that trauma is another factor which may enter definitely as one of the predisposing elements in atrophic arthritis. The degree of trauma that may be required to serve significantly as a predisposing influence varies greatly and is no doubt dependent upon other factors: the inherent susceptibility of the subject to arthritis, the existence of active foci of infection, the general constitutional state of the individual at the time the trauma is inflicted, and so on. From a purely clinical standpoint, experience alone would be sufficient to force acceptance of the conclusion that trauma is occasionally the last link in a chain of predispositional influences permitting the establishment of atrophic arthritis.

In an individual with a combination of inherited susceptibility and the stress of a sufficient number of such extraneous influences as have been discussed, we have a fully ripened candidate for atrophic arthritis. Although such an individual may be conscious from time to time of various types of neuralgic pains and aches, muscular fatigue, and stiffness, he is not yet arthritic; it requires still another factor—the seed—to precipitate the pathologic and clinical manifestations of frank arthritis.

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CHAPTER III

THE PRECIPITATING ETIOLOGIC FACTOR IN ATROPHIC ARTHRITIS

In the preceding section we discussed the predisposing factors in atrophic arthritis. What, then, is the precipitating factor? The writer holds the firm belief, shared by many serious students of this disease, that infection ultimately assumes a prominent role in precipitating the syndrome of atrophic arthritis. There are some dissenters from this view. On the whole, objections to the infectious etiology of atrophic arthritis represent a reaction to an exaggerated interpretation of the entire principle of focal infection, especially shortly after it was originally enunciated. Reasoning from the occasional direct association of infection and arthritis, there resulted, for a while, an overemphasis on the factor of infection, which overshadowed those many other significant attributes that we recognize today, and of which we have already spoken. It is the opinion of the writer that a wide view of the disease forces acceptance of the idea that both infection and an underlying physiologic disturbance participate in the production of the arthritic syndrome.

Let us now examine some of the evidence which leads one to accept some infective agent as the probable precipitating influence in atrophic arthritis.

BACTERIOLOGIC EVIDENCE

Shortly after the importance of bacteriology in relation to medicine was established, the search for the causative infective agent of atrophic arthritis was started. It is impossible to discuss at length, in a brief summary of this sort, the findings of many bacteriologic studies. *Nor is it important to do so.* It is enough to say, in passing, that the results obtained have been singularly contradictory. From time to time a great variety of organisms, isolated by various observers from joint tissues, regional lymph nodes, blood, and suspected foci of infection of patients with arthritis were described as the probable etiologic factor. Bacilli, diplococci, staphylococci, various types of streptococci, and diphtheroid bacilli have thus, at various times, been incriminated. Although the organisms that were recovered were

of a heterogeneous group streptococci predominated. Most observers were however unable to find any organisms in such cultures.

In 1936 McEwen, Alexander, and Bunim reported their study of the results of blood cultures in different forms of arthritis (including atrophic arthritis) and in normal cases used as controls. Incidentally, their paper includes a comprehensive review of the most recent bacteriologic studies on the blood in various forms of arthritis. They isolated streptococci chiefly green streptococci not only in a certain proportion of their cases of atrophic arthritis but also in certain cases suffering from diseases known to have other causes and from the blood of even normal persons. Lichtman and Gross too isolated such organisms almost as frequently from their control group of cases as from those with rheumatic fever and atrophic arthritis. For these reasons it has been concluded that the cause of atrophic arthritis cannot be ascribed to the organisms isolated in blood cultures from patients with the disease. It is apparent that under certain conditions of bacteriologic technique streptococci appear not only in the blood of persons with arthritis but also occasionally in that of normal persons particularly in that of individuals whose resistance has been lowered by illness.

Obviously the results of bacteriologic investigations of arthritic tissues and of the blood of arthritic patients are still in general, too contradictory to be accepted unequivocally as supporting the theory of infection as the basis of the disease. The author nevertheless feels that the isolation of bacteria—particularly of hemolytic streptococci—from the blood and joint tissues of patients with arthritis cannot be entirely ignored simply because control cultures also are occasionally positive for similar organisms.

We do not at present know how often such bacteremias occur normally and what their significance is. It is possible that such transient bacteremias in presumably normal persons are of some clinical importance. It may be that these bacterial migrations occur more frequently than we now suppose, that they are harmless so long as the basic resistance of the individual is adequate to offset their tendency to localize and produce disease. It is even possible that such bacteremias establish metastatic lesions in various organs including the joints and that these lesions remain smouldering at a subclinical level, without symptoms until the resistance is sufficiently lowered to permit the previously latent pathologic process to ripen into full blown disease.

Although we have so far, stressed the possible importance of streptococci in relation to rheumatic infection other bacteria may conceivably be concerned as well. Even attenuated tubercle bacilli have been suggested as the cause of typical atrophic arthritis. This view receives vague support from

some sources in England and on the Continent, but it cannot yet be regarded as more than an interesting assumption. However, the general idea that a variety of organisms may be concerned in typical atrophic arthritis cannot be dismissed.

That the apparent similarity of pathologic pictures does not necessarily imply bacteriologic unity of the etiologic agent is based on fact. The specific arthritides frequently associated with bacillary dysentery or infection by *Brucella abortus* present similar clinical manifestations, but have a widely different etiology. Cases of gonococcal origin, or those caused by the *Brucella abortus*, for example, could and often do masquerade as "non-specific atrophic arthritis" until search reveals the specific etiologic organism, identified by bacteriologic or serologic means. Clinically, such specific arthritides may be indistinguishable from the garden variety of atrophic arthritis, and so, while we are likely to emphasize the streptococcal relationship, the possibility of infection with other strains must not be dismissed. The evidence available at present would indicate, however, that from an etiologic standpoint streptococci may be of first importance in atrophic arthritis.

A VIRUS AS THE ETIOLOGIC AGENT

In 1935 Schlesinger and his co-workers presented the first careful experimental study hinting at the possibility of a virus as the cause of acute rheumatic fever. Since then other observations have been presented which deserve consideration. The report of Coles, and that of Eagles and his associates in England, confirmed in a large measure Schlesinger's conclusions that virus bodies may be isolated from various pathologic exudates both in acute rheumatism and in atrophic arthritis.

Recently the possibility of a virus bearing an etiologic relationship to atrophic arthritis and rheumatic fever has received further support. Sabin, at the Rockefeller Institute, isolated, from the brain of a mouse, a virus with which he consistently produced atrophic arthritis in mice by intravenous or intraperitoneal injection. Attempts at cultivating a filterable pleuropneumonia like virus from exudates and tissues of patients with atrophic arthritis or rheumatic fever have thus far proved unsuccessful.

The thought occurs, of course, that any virus proved to be etiologically related to human rheumatism would in one stroke explain why ordinary bacteriologic cultures have so frequently been found sterile. These possibilities, arising from purely hypothetical premises, are mentioned here merely to direct the attention of the reader to another path of research in the quest for the etiologic agent of rheumatic disease.

SEROLOGIC STUDIES

Serologic studies of the past few years have added more convincing evidence in favor of the streptococcal etiology of atrophic arthritis.

Nicholls and Stainsby were among the first to show the presence of agglutinins for streptococci in the blood of atrophic arthritics. They indicated that these agglutinins develop following the onset of the disease, reach a maximum during the height, diminish and finally disappear following recovery of the patient. Dawson, Olmstead and Boots later corroborated the presence of agglutinins for streptococci in extraordinarily high dilutions in the serum of patients suffering from atrophic arthritis although they had been unable to recover streptococci either from the blood or joints. They also found that the agglutinating principle in atrophic arthritis serum presented a certain degree of streptococcal group if not strain specificity. In 1936 McEwen, Bunim and Alexander confirmed the presence of agglutinins in a large proportion of their cases of atrophic arthritis.

That sera from the vast majority of cases of atrophic arthritis possess the capacity to agglutinate strains of hemolytic streptococci has been confirmed abundantly. This fact is now generally accepted. Because these agglutinins are not strain specific and because of other evidence which we cannot discuss here, the exact interpretation of the agglutination reaction of arthritic sera to hemolytic streptococci cannot yet be given. Most of the evidence indicates, however, that so definite a serologic reaction in so large a proportion of patients with atrophic arthritis is more than suggestive of the probability that hemolytic streptococci are in some way concerned in this type of arthritic process.

PRECIPITINS

Dawson, Olmstead and Jost also investigated the sera of such patients for precipitins against various protein and carbohydrate fractions of hemolytic streptococci. Although they could not establish an absolute agreement, they found a close approximation between the capacity of atrophic arthritis serum to agglutinate strains of streptococcus hemolyticus and to precipitate group-specific fractions of the same organism. McEwen, Bunim and Alexander confirmed the presence of precipitins in the blood of patients with atrophic arthritis but pointed out that positive precipitation reactions were occasionally observed also in patients with other conditions and even at times in supposedly normal individuals.

These serologic findings supporting as they do in a large measure, the bacteriologic data cited, lend substance to the theory that the presence of

streptococci is in some way related to a large proportion of the cases of atrophic arthritis. Whether these organisms play a primary etiologic role or whether they are merely secondary invaders is yet to be decided.

ANTISTREPTOLYSIN

Iodd has shown that the streptococcal hemolysin is antigenic, stimulating the production of antibody (antistreptolysin) which may be found in high titers in acute infections with hemolytic streptococci. Since Iodd's original observation in 1932, others have studied the antistreptolysin content of the sera of patients with atrophic arthritis. Some, notably Blair and Hallman, and Griffiths, have noted antistreptolysin titers definitely above normal in a considerable proportion of patients with atrophic arthritis. Others have found the antistreptolysin content normal in similar cases. It appears that the antistreptolysin titer is higher in early acute cases than it is in more advanced, chronic cases. This may explain the normal antistreptolysin content of certain arthritis sera studied. In any event, the high antistreptolysin titers in certain groups of atrophic arthritis cases cannot be entirely dismissed. They substantiate the theory that the hemolytic streptococcus is probably a factor in the syndrome of atrophic arthritis.

THE PATHOLOGIC PICTURE

The pathologic changes in the joint tissues of atrophic arthritis, including as they do all the signs of an exudative inflammatory process, with cellular infiltration and necrosis, add still more evidence that infection may be an etiologic factor. Such pathologic changes contrast strikingly with the histologic picture found in hypertrophic (osteo-) arthritis. In the latter we find not an exudative inflammatory reaction, but a bland, predominantly degenerative pathologic process, consistent with its assumed noninfectious origin.

FOCAL INFECTION

The etiologic relationship of focal sepsis to atrophic arthritis, though subject to controversy even today, is definitely accepted by most clinicians. There is evidence that focal infection occurs more frequently and plays a more active part in atrophic arthritis than in any other form of chronic joint disease. The subject is so important in relation to the pathogenesis of arthritis that it will be discussed more fully in a separate section later. We mention it here merely as a reminder of another segment of evidence in favor of the infective nature of atrophic arthritis.

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EXPERIMENTAL REPRODUCTION OF THE DISEASE

Recently Cecil and Angevine have produced a chronic (nonsuppurative) proliferative arthritis in rabbits by injecting small doses of strains of hemolytic streptococci and streptococcus viridans. The pathologic lesion thus induced bore a striking similarity to that of atrophic arthritis in man. Obviously the clinical syndrome of atrophic arthritis cannot be reproduced experimentally because as we have already said the clinical manifestations of the disease in man are probably dependent on more than mere infection. Nevertheless the pathologic picture of the arthritis so induced lends additional weight to the argument that infection is in some way related to atrophic arthritis.

THE CLINICAL EVIDENCE

There are still other indications that infection enters in some manner in the syndrome of atrophic arthritis. At least in certain obvious acute infections such as scarlet fever, typhoid fever, brucellosis, ulcerative colitis, and so on, there is the frequent occurrence of arthritis in the course of the disease. Again the sedimentation rate of the red blood cells is definitely increased during the active stages of atrophic arthritis, and the degree of this increase is fairly proportional to the activity or severity of the process. Moreover any fundamental change in the clinical condition is generally reflected in immediate corresponding change in the sedimentation rate. On the other hand in hypertrophic (osteo) arthritis, where everything points to a noninfectious degenerative process as the cause, the rate of sedimentation of erythrocytes is most often perfectly normal unless superimposed infection or extensive alteration of the joint has occurred.

It is recognized that the Schilling hemogram is a very sensitive indicator of the degree of activity in any infectious process. In atrophic arthritis a considerable percentage of the cases show a definite increase in the proportion of young polymorphonuclear leucocytes—the so-called shift to the left.

There is no denying the importance of precise bacteriologic and serologic data in support of the idea that infection enters in some manner in the syndrome of atrophic arthritis. Actually, however, nothing is today so convincing as the clinical manifestations of the disease. The condition of the patient with atrophic arthritis, particularly when the disease is active, presents many features pointing to infection as a cause. The general debility, the slight fever occasionally observed, a somewhat rapid pulse, poor appetite, disturbed nutrition with weight loss, the frequent occurrence of

various grades of anemia—all these suggest a low grade, smouldering infection. They indicate, too, that infection is not confined to the joints alone, but produces its effects on the body as a whole.

These systemic manifestations of atrophic arthritis are, indeed, the most distinguishing features of this form of rheumatism, they are not observed in patients who have other types, as hypertrophic arthritis. Although the changes in the joints themselves vary from those seen in other forms of rheumatism, these differences are less sharp than the signs of general infection so clearly discernible in the patient with atrophic arthritis.

To be sure, infection alone is not the beginning nor the end, of atrophic arthritis. Many inherent factors in the patient, combined with certain extraneous influences plus infection do, however, explain the rheumatoid syndrome. It might be argued that such a systemic infection results from, or follows the arthritic disease but is not causally related to it. The course of events does not support this idea. We must admit that the case for the infectious etiology of this disease is weakened by certain contradictory findings in the bacteriologic and serologic investigations. We accept the fact that unequivocal proof of the infectious etiology of atrophic arthritis is still to be presented. But, from the clinical point of view, we are forced to take a firm stand, one which will offer guidance to the therapeutic approach. From that standpoint we must rely on the evidence now at hand. Doing so, the author is inevitably led to accept infection as the most likely precipitating factor in rheumatoid arthritis, and, from the standpoint of the practicing physician, finds no theory of pathogenesis so logical or so helpful in the actual management of the disease.

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CHAPTER IV

PATHOLOGY OF ATROPHIC ARTHRITIS

The pathologic changes in atrophic arthritis vary with the severity of the process and the stage of the disease. In most cases the synovial membrane is probably the earliest affected. It becomes edematous and congested showing grayish red or purple, if the vascularity and congestion are very pronounced. It may become thickened and may proliferate into tags of inflammatory tissue, forming a vascular 'pannus' which projects into the joint cavity. Some of these tags adhere to the joint cartilage, which becomes pitted, necrotic, and gradually eroded. The surrounding cartilage attempts repair of this damage, should the necrosis and erosion proceed faster than the rate of repair, the entire thickness of the cartilage may be penetrated down to the cancellous, epiphyseal end of the bone.

Microscopically, there is evidence of edema both within the cells of the synovial lining and within the interstitial areolar tissue. Generally hyperplasia occurs, so that, instead of a single layer of cells, as is found normally, several layers constitute the synovial lining.

Such cellular hyperplasia may be most pronounced in the villi. There is a rich blood vascular network in the loose interstitial tissue just beneath the lining cells and extending into the villi. Scattered throughout the lining membrane are numerous exudative cells, chiefly lymphocytes, monocytes, and occasional polymorphonuclear leucocytes (Figs. 4, 5). The more acute the process the more numerous are the polymorphonuclear cells, however, they seldom predominate. The lymphocytes constitute the predominant cell types even in active cases, decreasing in number as the proportion of monocytes increases with advancing chronicity. Here and there these cells may form more closely packed aggregations, sometimes appearing as rounded masses. In some instances capillaries pierce the centers of such focal collections of cells. But as Ghormley and Deacon (1936) have shown, these are not necessarily perivascular. In older cases, varying degrees of fibrosis may be noted.

Naturally, the microscopic picture varies in different sections of synovia of any given joint. In one area cellular exudation and active proliferation of membrane cells and capillaries are evident, in another the exudative cells and capillaries are much less numerous and fibrosis more pronounced. These

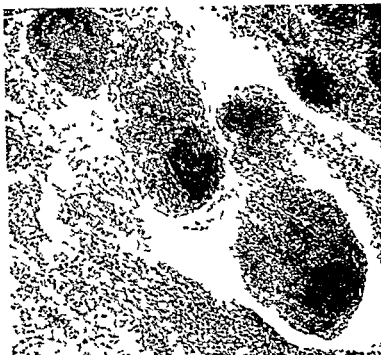


FIG. 4 Section of synovia and villi from interphalangeal joint of a finger in atrophic arthritis showing diffuse cellular infiltration (Gram stain $\times 100$)

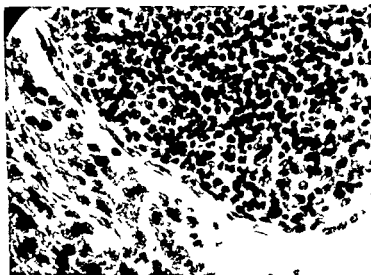


FIG. 5 Higher magnification of synovia illustrated above revealing infiltration of tissue with lymphocytes plasma cells and monocytes (Gram stain $\times 400$)

variations probably reflect the stage of activity of the pathologic process and its duration

Another coincident focus of inflammatory reaction may be set up in the



FIG 6 Thinning of bone trabeculae and focal collections of lymphocytes in the marrow spaces of epiphysis adjacent to affected joint in atrophic arthritis ($\times 50$) (From R K Ghormley, *The Pathology of Non Specific Arthritis In A Survey of Chronic Rheumatic Diseases* Oxford Press, 1938)

epiphyseal marrow, just beneath the cartilage. Focal collections of monocytes, increased vascularity of the epiphyseal marrow, and proliferation of connective tissue may constitute a prominent feature of the pathologic process. This inflammatory process may burrow its way toward the cavity of the joint. The bony trabeculae in the epiphyseal ends of the bones, adjacent to affected joints, are attenuated, there is also some degree of general decalcification at the articular ends of the bones. The thinning of the bony trabeculae may represent, in part at least, a result of the proliferative reaction within the epiphyseal marrow (Fig 6).

The fibrous capsule usually is involved in the inflammatory process, becoming edematous and, later, thickened through fibrosis. Partly from dis

use and partly from direct inflammatory changes the muscles undergo varying degrees of atrophy.

Destruction of the joint cartilage appears to be largely the result of the



FIG. 6. Inflammatory pannus arising from epiphysis invading the deeper layers of the joint cartilage. By progression of such a process the cartilage may be penetrated the subchondral granulations extending directly into the joint cavity ($\times 50$). (From R. K. Ghormley, *The Pathology of Non Specific Arthritis In A Survey of Chronic Rheumatic Diseases*, Oxford Press, 1938.)

inflammatory process in the surrounding structures. We have already indicated that as the synovial pannus extends from the margin of the joint toward the center it becomes attached to the cartilage gradually eroding it. And we have also indicated that some of the villi actually burrow their way through the full depth of the cartilage projecting into the inflammatory mass in the subchondral epiphyseal marrow. In a similar manner the inflammatory process in the epiphysis may destroy the cartilage from beneath penetrating it and extending directly toward the synovial pannus (Fig. 7).

Such changes in the synovia, the cartilage, the epiphysis, and the capsule develop step by step. Sometimes one area of inflammation lags, or its extension is halted entirely, and the joint recovers before much damage has been done. On the other hand, if the progress of the inflammation is not impeded, the cartilage gradually disappears and the joint cavity becomes filled with a soft, spongy mass of inflammatory tissue which has attached to the raw and roughened ends of the bones constituting the joint.

The synovial fluid is generally increased in amount large effusions sometimes developing and distending the joint capsule In such cases the fluid appears more or less turbid but is never purulent

As the activity of the process declines the edema recedes Proliferation of connective tissue replaces the exudative cellular reaction If damage to the joint tissues is not too extensive repair ensues and the joint is reconstructed fairly well although it may retain many battle scars which in turn may interfere slightly with motion Contraction of the muscles by spasm or scar tissue may limit motion still further

If the damage to joint tissues has been more extensive all the inflammatory tissue is converted into a mass of scar tissue binding the bones together Gradually the binding scar tissue contracts becoming more solid and more like cartilage Later lime salts are deposited and the joint is replaced by a rigid mass of bone This is ankylosis The joint is thereby deformed and incapable of further motion The character of the deformity at this time depends on the position in which the bones were kept during the healing process and that in turn depends on the pulling effect of the muscles which maintained the greatest power

Nor is the damage manifest in the joints alone In mild cases the visceral effects may be scarcely perceptible in more severe cases the damage is extensive The liver the spleen and the bone marrow are probably always affected to a greater or lesser extent because normally those tissues are the depositories for the debris of infection Involvement of the reticulo-endothelial system may account for the anemia which occurs frequently in this type of arthritis

Certain liver function tests have revealed evidence of varying degrees of liver damage in a high proportion of patients with atrophic arthritis (Rawls 1939) It is doubtful whether such disturbances of hepatic function play any very important role in the disease It appears more likely that the liver is among other organs merely caught up in the diffuse pathologic process which affects so many tissues

We find most striking evidence of liver and spleen involvement in children who suffer from atrophic arthritis (Still's disease) They may present huge enlargement of the liver and spleen and hyperplasia of lymph nodes associated with arthritis and anemia We used to regard Still's disease as a distinct variety of atrophic arthritis now we believe it to be only a variation from the common variety of atrophic arthritis The striking lymphadenopathy liver and spleen enlargement and anemia are peculiar pathologic reactions possibly conditioned by the age of the patient However Felty has described a similar syndrome in adults

The subcutaneous nodules of atrophic arthritis present many clinical and pathologic similarities to those which occur in rheumatic fever Although

Collins has pointed out differences in the pathologic picture of the subcutaneous nodule in acute rheumatic fever and atrophic arthritis, there is much evidence (particularly that presented by Dawson) that the nodules in both of these conditions are essentially alike. Whatever differences may exist are probably differences of degree and not of kind. It is likely, too, that the pathologic picture of the nodule of atrophic arthritis is modified to an extent by trauma from pressure.

Rheumatic lesions of the heart valves, pericardium, or heart muscle occur rarely in typical atrophic arthritis. The tendency to heart involvement is greater if the onset of the disease occurs before the age of thirty. When rheumatic heart disease does develop in the course of atrophic arthritis the lesions in the heart are essentially like those which occur in acute rheumatic carditis.

Serum protein content is significantly altered in atrophic arthritis. There is a lowering of the plasma albumin in this condition and an increase in plasma globulin. There is then a reduction of the albumin globulin ratio. Such changes in the serum proteins do not occur in hypertrophic arthritis (Davis 1936; Scull, et al. 1939). These abnormalities may be related and to an extent explain the rapid rate of erythrocyte sedimentation in atrophic arthritis. Such changes in the protein content of the blood also add indirect evidence that infection is an important etiologic mechanism in this type of arthritis. As Pemberton indicated, such changes suggest the necessity for the removal of various antigenic substances (as infectious foci) which have a tendency to stimulate globulin production. These changes also indicate the need for protecting joints against undue damage, stemming thus the amounts of "foreign protein" which, if liberated into the circulation, would interfere with albumin production and stimulate that of globulin. Perhaps most important of all, the lowered albumin concentration in the serum indicates clearly the necessity for the provision of an ample intake of protein—not a curtailment of it, as was at one time urged.

Closely linked with these deviations in protein metabolism are the findings of Scull and Pemberton. They found that in some cases of atrophic arthritis a peripheral edema of the tissues occurs which is not primarily due to simple circulatory stasis. Such an edema may well be related to a reduced colloid osmotic tension of the blood, caused by the decreased albumin concentration in the serum, a situation analogous to that which occurs on a larger scale in lipoid nephrosis.

The basal metabolic rate is significantly, though only slightly, lowered in some cases of atrophic arthritis. In many of them it is normal. There is no indication that the lowered rate, when it exists, is primarily the result of thyroid underactivity. The evidence available at present suggests rather that the decreased rate of metabolism is merely an expression of the diffusely

slowed metabolic activity which frequently characterizes the rheumatoid state. This may in turn be dependent upon the sluggish circulatory flow which is a manifestation of this type of arthritis. A similar explanation may apply to the delayed rate of sugar removal from the blood which Pemberton showed may exist in patients with this disease.

All of us are familiar with the loose reference to the calcium and parathyroid disturbance in arthritis. However, no convincing proof has been presented that such a disturbance exists. Neither clinical nor pathologic evidence of parathyroid disease has ever been demonstrated. In all reliable investigations the blood calcium concentration has been found entirely normal. Because decalcification and osteoporosis develop to some extent in many cases of atrophic arthritis suspicion was naturally focused on the calcium metabolism and parathyroids. Whatever the manner of production of such osteoporosis it is not primarily a disturbance in either parathyroid metabolism or the metabolism of calcium in the generally accepted meaning of those terms.

Nor is there evidence of any disturbance in the uric acid metabolism in atrophic arthritis. The nonprotein nitrogen concentration in the blood is as a rule not affected. Disease of the kidneys and disturbances of renal function are very unusual. We have encountered isolated instances in which a pronounced focal nephritis or glomerulonephritis coexisted. Although the renal disease and the arthritis might have been linked to an etiologic mechanism common to both there is no indication whatsoever that the renal disease plays any pathogenetic role in the arthritic syndrome. The blood urea or uric acid concentration or both may show some elevation when such concomitant renal disease exists. The correct interpretation of these findings indicates however that they result directly from impairment of renal function not from any metabolic deviation characteristic of the arthritic syndrome.

There is no demonstrable disturbance in the concentration of the more important blood electrolytes.

The total cholesterol content of the plasma tends to be slightly decreased in atrophic arthritis (Hartung and Bruger). The more active the arthritis the more pronounced is the plasma cholesterol deficiency. This finding contrasts with the slight elevation in the plasma cholesterol in hypertrophic arthritis. Since acute infectious processes are generally accompanied by hypocholesterolemia the reduction of the plasma cholesterol in active atrophic arthritis may be regarded as not of basic importance.

Contrary to previous presumptions the recent work of Freyberg establishes beyond doubt that a disturbance of sulphur metabolism does not exist in atrophic arthritis.

The peripheral circulation is affected to some extent in practically all

cases. This circulatory disturbance is evident clinically in the cold hands and feet, the tendency to pale or cyanotic fingernails, paresthesias, and in the relief afforded such arthritic joints by the use of heat.



FIG. 8. Marked flexion and hyperextension deformities of the fingers, with ankylosis, in a case of advanced atrophic arthritis in which practically every joint was affected. Note the glossy taut skin which has a distinctly sclerodermatous character, the scleroderma developed long after the arthritis.

The important relationship between vasospasm and the arthritic process is evident in at least certain cases in which typical atrophic arthritis is accompanied by scleroderma, sometimes of a very marked degree (Fig. 8). In this connection we do not refer to primary scleroderma with secondary joint changes but rather to those cases of primary, widespread atrophic arthritis with marked destruction of joints, and frequently ankylosis, in which scleroderma supervenes. Such patients generally present a history of vasomotor phenomena of a vasospastic type affecting the extremities. These changes may date back many years, indicating the existence of a vasospastic diathesis preceding the development of the arthritis. Although such an association between atrophic arthritis and scleroderma is not commonly observed, it appears often enough to suggest that a vasospastic factor may enter into the process in many arthritic patients, although it produces profound disturbances in only a few.

Numerous experiments have established that relative constriction of the peripheral capillary bed, apparently caused by functional vasospasm, exists in arthritis. This circulatory abnormality is much more pronounced in atrophic arthritis than in any other type or in any other pathologic condition, except Raynaud's disease. This deficiency in the peripheral circulation is not limited to the surface tissues, as the skin, alone. It has been shown that similar circulatory impoverishment probably exists also in the deeper structures of the joint—the synovia, periarticular capsule, and bones.

The tendency to constriction of the peripheral circulation in arthritis may

be to a large extent an inherited defect, dependent upon a peculiar physiologic action of the autonomic nervous system. The circulatory bed may, however, be additionally embarrassed by such factors as fatigue, infection, nervous anxiety, poor posture, and climatic influences.

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CHAPTER V

PATHOGENESIS OF ATROPHIC ARTHRITIS

The manner in which infection might operate to produce the manifestations of atrophic arthritis is not entirely clear. The occurrence of bacteremia and the cultivation of bacteria from joint tissues might point to actual dissemination of these organisms from certain foci of infection and to their lodgment in various parts about the joint. The pathologic changes might then be regarded as an ordinary inflammatory reaction to bacterial invasion with necrosis and varying degrees of repair leading to the sequelae of the disease.

As we have stated in a previous section question arises as to why the causative organisms are not isolated more frequently and more consistently from the suspected causative foci of infection the blood and the joint tissues. It is possible that the bacteremia in arthritis is a variable mild transitory state not always amenable to bacteriologic demonstration. Cecil and Angevine producing a known infective arthritis in rabbits by the intravenous injection of hemolytic streptococci and streptococcus viridans were seldom able to recover the organism from the blood after the third or fourth day even when changes in the joints persisted for many months. Our experience was identical in even more acute forms of experimentally induced streptococcal arthritis. It is conceivable then that even if a state of bacteremia intervenes in the establishment of metastatic joint lesions cultures of the blood are not necessarily consistently positive unless a constant influx of organisms occurs which is unlikely.

Bacteriologic cultures of joint tissues are not resorted to in the early active stages of the inflammatory process because at such times tissue is not easily available for study. Joint tissues generally become available for culture only in the late quiescent or inactive stages of the disease that is during operations for clearing the debris of previous joint destruction.

Synovial fluid which is available at almost any stage of the disease is frequently bacteria free on culture. This state can be explained by the fact that the pathologic lesion of active arthritis is predominantly one of the synovial membrane articular cartilage and epiphyseal bone marrow, the exudation of fluid into the joint is merely a reaction to the inflammatory

process in the synovia. Therefore, any bacteria present in the synovial fluid may represent merely an overflow from inflammatory foci in the vicinity.

In this connection, our experience with cytologic studies and blood cultures in experimental arthritis, induced by intravenous injection of streptococcus viridans, has been particularly instructive. In numerous such experiments, performed in 1930, we were impressed by the rapidity with which the synovial fluid rid itself of bacteria which invade it shortly after infection of the joint occurs. During the first few days of the acute arthritic process we found the fluid quite turbid, as a result of massive infiltration with polymorphonuclear leucocytes and fibrin, and at such times the organism injected could generally be isolated from the synovial fluid in pure culture. After the third or fourth day, however, the fluid became less turbid in appearance, the polymorphonuclear leucocytes were gradually replaced by progressively larger proportions of phagocytic mononuclear cells, and recovery of the injected organism became more difficult. By the seventh to tenth day, cultures of the synovial fluid became sterile although there was still considerable evidence of inflammation in the synovia and periarthicular structures.

Cecil and Angevine described an almost identical course of events. First producing a low grade, chronic proliferative arthritis in their experimental animals, they were able to culture streptococci repeatedly from aspirated synovial fluid during the first week, but the organism could rarely be recovered from the synovial fluid and tissues after the third week, even when changes in the joints persisted for as long as eighteen months.

These experimental findings indicate that if infection enters into the pathogenesis of atrophic arthritis, its mechanism of action as a metastatic infection from foci, through the blood stream, to joints, is not unlikely, even though bacteriologic studies do not always substantiate this idea.

The possibility that a filterable virus, perhaps even related to the streptococcus, enters etiologically in relation to rheumatoid arthritis has already been discussed (page 37).

BACTERIAL ALLERGY IN THE PATHOGENESIS

It has been suggested that besides the ordinary inflammatory reaction to direct infection in the joints, the factor of allergic sensitization to the bacteria should be considered—a factor, it is contended, important in maintaining clinical activity of the disease. Proponents of the latter view explain that perpetuation of the distant disease, or its periodic recurrence, depends upon the maintenance of an allergic reaction between the products of the bacteria in some focus of infection and the sensitized tissues elsewhere.

Although this is a possibility, there is not yet any proof that allergy, in

the sense in which we generally think of it, can be invoked to explain all the pathologic and clinical phenomena of the arthritic state. In a critical analysis of this concept Wolf said: "It seems as though the whole theory of allergy has been introduced as the result of a desire to explain something which it would be far more satisfactory to describe. The conception of allergy does not explain rheumatic and arthritic manifestations in general. It does not help us in therapeutics. It really only complicates the problem. The whole theory itself is vague and not clear enough to be used to clear up other problems. And this agrees with the conclusion of Freeman, to whom the word allergy is not a gleam of sunshine breaking through, but an extra wisp of fog. It is none the less possible that the initial injury to joints by infective elements in whatever manner achieved, makes such articulations more sensitive to additional assaults from relatively infinitesimal quantities of bacteria or their products. That is not allergy in the strictest sense of that word. But such a hypothesis would explain exacerbations of existing arthritis by upper respiratory infections, by manipulation of suspected foci, as during operations for their removal, and like circumstances."

BACTERIAL TOXINS

There are still other theories invoking still other etiologic factors as being the important precipitating influence in atrophic arthritis. The idea that arthritis may be produced by toxins liberated from organisms in foci of infection has received some attention, but no convincing proof of the theory has been produced.

METABOLIC AND ENDOCRINE ABNORMALITIES

The clinical picture generally associated with systemic infections may occasionally closely resemble acute exacerbations of certain metabolic states such as diabetic coma, uremia, gout, and so forth. It is hardly likely, however, that an analogous metabolic upset can be at the bottom of the rheumatoid syndrome without revealing itself in any significant deviation of metabolism that could be detected clinically.

It is difficult to reconcile the clinical manifestations of the disease and the facts already cited with the view that arthritis is primarily a disease of disturbed metabolism, or dependent on endocrine or neurogenic abnormalities. Detailed discussion of these aspects of the subject would lead to a figurative jungle of theories in which no trail has yet been blazed for the practicing physician.

Although atrophic arthritis may be accompanied by profound metabolic derangement, no proof exists that a metabolic dyscrasia is the mainspring of the arthritis. A more logical view is that the metabolic disturbance is secondary to some other factor, the same view applies to associated endocrine and neurogenic abnormalities. Some endocrine abnormalities are probably purely incidental to the disease, others may contribute indirectly in one way or another. The author has observed a number of instances of hyperthyroidism associated in various chronological relationships with atrophic arthritis. In no instance was a direct etiologic relationship between the two diseases indicated. In some of these cases the hyperthyroidism no doubt imposed an additional and difficult load on the arthritis patient but it is significant that in no instance did thyroidectomy contribute evidently to alleviation of the arthritic process. In several instances the arthritis progressed despite cure of the hyperthyroidism. According to Edgecombe there is even less proof of any relationship between other endocrine abnormalities and atrophic arthritis.

NUTRITIONAL DISTURBANCES AND VITAMIN DEFICIENCY

Because there is striking evidence of nutritional abnormalities, some writers have assumed that atrophic arthritis is primarily a disturbance in the "metabolism of nutrition." We do not deny the importance of nutritional abnormalities, but neither do we find conclusive proof of their playing more than a contributory role in the pathogenesis of this disease.

We have already indicated (page 27) that deficiency of vitamin B has long been suspected as a factor in the pathogenesis of atrophic arthritis. The evidence supporting this idea may be largely coincidental, though, from a clinical standpoint, quite impressive. The changes in the mucous membrane of the tongue, the atrophy of the mucosa, and the occasional glossitis observed, as well as the changes in the configuration and function of the colon, suggest that vitamin B deficiency very likely enters into the rheumatoid process. The glossitis may be marked to the point of ulceration. When these changes are associated with a brownish pigmentation of the hands, feet, and face, the clinical impression is that of a pellagra like syndrome. Since these manifestations sometimes respond favorably and promptly to the administration of nicotinic acid, clinical suspicion of the existence of latent pellagra in some cases of atrophic arthritis is, we think, justifiable. In other instances we have noted fairly conclusive evidence of predominantly riboflavin deficiency.

Deficiency of vitamins A and D has also been suspected, but never well established.

There is little question that vitamin C deficiency exists in atrophic

arthritis as in rheumatic fever Rinehart and his associates have shown consistently low (in many cases very low) values for the concentration of ascorbic acid (vitamin C) in the blood plasma of atrophic arthritis patients. Furthermore, it is extremely difficult to restore a normal plasma level of ascorbic acid even when relatively large amounts of vitamin C are administered. Such a disturbance has not been found to be a consistent feature of hypertrophic arthritis. Rinehart has also demonstrated experimentally the importance of vitamin C. Animals deprived of ascorbic acid became highly susceptible to the injection of bacterial cultures and developed rather typical infectious arthritis. Neither vitamin C deficiency nor the bacteria, alone, was capable of producing the characteristic arthritic changes.

An interesting recent study by Kaiser and Flavin indicates the possibility that the content of vitamin C in both the blood and tonsils of rheumatic children, might have some bearing on the incidence of invasion of the tonsils by hemolytic streptococci and on the virulence of these invaders. They found that the vitamin C content of the blood or tonsils was generally lower in children with tonsils containing hemolytic streptococci than in those with tonsils either not containing the organisms or containing organisms totally avirulent or of relatively low degrees of virulence. Here is evidence of one tangible factor which may operate in the maintenance of that balance we call normal resistance. Such a factor might also determine to some extent the variable degree of activity of a given focus of infection at different times.

All this evidence indicates that vitamin C deficiency is certainly part of the physiologic disturbance demonstrable in atrophic arthritis. The specific pathogenetic relationship of vitamin C deficiency in this condition is not yet clear, however. Low levels of ascorbic acid in the blood plasma are not peculiar to atrophic arthritis; they occur in nutritional deficiency states without arthritis. There is furthermore a distinct tendency toward vitamin C deficiency in the course of a great variety of infectious states.

What, then, is the place that should be assigned to vitamin deficiencies in general in the pathogenesis of atrophic arthritis? Are these changes responsible to some extent for the onset of the disease? Do they result from some metabolic or nutritional defect that results from the arthritis? Or is such nutritional deficiency merely a concomitant of the widely disturbed physiologic functions in this disease? The answer to these questions is important, unfortunately, it is not yet available.

NEUROGENIC ABNORMALITIES

We have already intimated that nervous influences of diverse types—both functional and organic—enter into some cases of atrophic arthritis but not

into others. This fact would indicate that neurogenic abnormalities are not of primary importance. It is likely that where they occur they contribute to disturbance of physiologic balance and thereby aid other factors more responsible for initiating the arthritic process.

Obviously, the influence of the numerous etiologic factors varies with different cases. It is conceivable for example that a patient, highly susceptible to atrophic arthritis because of a marked inherent tendency, may develop the disease when exposed to relatively minor degrees of systemic infection, especially if he is subjected to stresses and strains which further lower his resistance. On the other hand another individual, relatively better fortified by heredity may for many years escape severe systemic intoxication from infection which he may harbor. When arthritis develops in such an individual it may be the result of malnutrition or vitamin deficiency, or even of an acute endocrine disorder such as hyperthyroidism. The nutritional or endocrine dyscrasia may be prominent in the resulting clinical picture though the factor of infection may be more directly related to the pathologic condition in the joints. In view of this possibility it is essential that all factors be accorded the importance due them.

Clinically it is of practical value to attempt to appraise in what proportion various etiologic factors are operating in a given case. We have no precise technique for measuring these proportions, but conscientious estimates may serve us well.

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[For a list of books and monographs dealing with the general aspects of
chronic rheumatic disease (including considerations on the subject of the
present chapter) see page 527]

*Bacteriologic Data Relating to the Possible Mechanism of the
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See bibliography Chapter III (page 42)

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CHAPTER VI

FOCAL INFECTION IN ATROPHIC ARTHRITIS

GENERAL CONSIDERATIONS

In the present discussion we employ the term focal infection as denoting a systemic reaction to absorption of bacteria or their toxic products from localized areas of chronic infection. However this does not necessarily incriminate the areas of localized sepsis as a cause of disease. It is common knowledge that the presence of even pathogenic micro organisms in many situations is compatible with normal health. Similarly areas of localized infection whether a pustule in the skin or a dental periapical abscess are only potential sources of trouble foci of infection pathologically but not necessarily clinically. Such a distinction in the meaning of the term is necessary to offset the impression that a focus of infection is as someone has said merely anything that is readily accessible to surgery.

Areas of focal sepsis exist in many patients without evidence of arthritis. It is illogical however to assume from this that there is no relationship between focal infection and atrophic arthritis. Gonococcal arthritis when it occurs is always secondary to a gonococcal focus of infection though many patients with active gonococcal foci of infection never develop arthritis.

Focal infection must therefore be a pathologic state which comes into play only when there is disturbance between those relative qualities of individual resistance and bacterial virulence. It follows that the clinician must take this into consideration and must in turn recognize that the element of human resistance to infection is conditioned by many factors. This resistance undoubtedly overcomes the bacterial organism in those cases where the individual seems unaffected though infectious foci exist. In other words even though the bacteria may be transported to distant organs from time to time they cannot become implanted due to this resistance.

In the early stages of typhoid fever the blood is invaded by the typhoid bacillus in nearly every instance yet localization remains predominantly in lymphoid structures the pneumococcus invades the blood in lobar pneumonia yet the lesions are predominantly in the lungs and pleura in chronic septic endocarditis the presence of streptococcus viridans may be

demonstrated in the blood daily or at intervals for many weeks yet in most instances localization remains limited to the valves of the heart. What then influences the localization of the bacteria? For such widely varying types of bacteria as those just mentioned one may presume a peculiar specificity of the bacterial body for different tissues. But in the case of focal infection as that term is usually employed the systemic effects are variable in the extreme though the organisms concerned are predominantly streptococci. In some cases mitis results in others endocarditis in still others arthritis or neuritis. How then are we to explain the multiplicity of type of metastatic lesions from a group of organisms so closely alike?

Consideration of the various streptococci on the basis of certain of their biologic or morphologic features has resulted in classification of most of them into several arbitrary groups. Nevertheless we cannot associate any one group with any particular pathologic lesion. The streptococci isolated in various cases of endocarditis have not for example always been of the same group. Similarly the streptococci isolated by various observers from patients with arthritis have shown wide variation in their biologic reactions to various sugars and in their production of pigment on blood agar.

Although the ordinary cultural characteristics of the various types of streptococci found in atrophic arthritis have varied widely their actual type specificity so far as their proclivity to produce a specific pathologic lesion is concerned may possibly be quite fixed. Thus is presented the further possibility that the various types of streptococci are actually from the standpoint of their pathogenicity of one specific type though seemingly unrelated culturally.

In this connection mention must be made of the concept of bacterial mutation which recognizes wide variability in cultural characteristics of many organisms in various phases of their life cycle. Such mutation has been demonstrated among the streptococci as among other bacteria. Under certain cultural conditions hemolytic streptococci may for example change into forms which do not hemolyze blood but which have the capacity to produce green pigment on blood agar (characteristics of the streptococcus viridans). Short chained streptococci have been shown to change to diphtheroid forms under proper conditions reversal of this mutation may be effected. These observations lead one to suspect that the seeming variability in the type of streptococci in any given disease may be only mutant not specific. It is thus possible that the various streptococci found in atrophic arthritis are mutant forms of one species of organism with a rather specific pathogenicity characteristic for the group as a whole.

As long ago as 1819 Benjamin Rush was impressed by the possible relationship between dental infection and arthritis. However the relationship of certain circumscribed areas of infection to chronic joint disease became

apparent only in recent years. This relationship was established by Billings who in 1912 recorded his now classic observations which pointed to the principle that focal infection in various sites particularly in the tonsils could be etiologically related to chronic arthritis. In the following year he reaffirmed this possibility. He pointed out that while the most common site of focal sepsis was the tonsils the teeth or sinuses it was occasionally in the prostate gland seminal vesicles and the generative tract in the female. He also intimated that it could probably exist as chronic appendicitis or cholecystitis or as a localized streptococcus infection anywhere.

Stimulated by Billings' clinical observations Rosenow arrived at the conclusion that a relationship exists between atrophic arthritis and streptococci of low virulence such as are found in foci of infection. He based his conclusion on the following facts. Streptococci isolated from foci of infection when injected into rabbits have a preponderant selective localizing capacity for the joints of the experimental animal where they set up an arthritis from such experimentally induced arthritic lesions the organism may be isolated in pure culture organisms from foci of infection of patients suffering from other diseases have much less propensity to produce experimental joint lesions on animal injection.

While this conclusion of Rosenow has not passed unchallenged it offers the most plausible explanation so far advanced for the mechanism of focal infection. A tentative acceptance of Rosenow's views is rendered justifiable by voluminous confirmatory evidence adduced by many of his co-workers and by other independent investigators.

In more recent years Rosenow and his associates have demonstrated the pathogenic specificity of various strains of streptococci by measuring their cataphoretic mobility that is essentially their rate of movement across an electrically charged field.

It has long been known that bacteria are colloids bearing negative electrical charges which can be measured in an appropriate apparatus. The interesting observations along this line may be summarized as follows. From patients with any given disease suspected of being caused by focal infection obtained strains of streptococci exhibited preponderantly a given speed of mobility (cataphoretic velocity) which varied distinctly from that of other strains isolated from normal controls and from patients with other conditions. In other words the grouping of strains of streptococci according to their cataphoretic velocity corresponds in general with the type of lesions which they produced.

Although application clinically is at present limited the scientific data just mentioned are too impressive to be ignored.

There has been much controversy and diverse interpretation of the concept of focal infection since Billings outlined it. The pendulum has swung

between relative conservatism and extreme radicalism in its clinical application. Owing to the fact that clinical distinction between atrophic and hypertrophic arthritis was not attempted to any extent until recent years even conservative clinicians were a little too prone perhaps to remove whatever foci were discovered in such patients. By and large the idea of focal sepsis in relation to arthritis was seized upon with more enthusiasm than judgment thereby inviting as a normal response an attitude of extreme scepticism concerning its significance. Every practitioner has seen the victims of such unharnessed enthusiasm. Misdirected efforts to eradicate or cure atrophic arthritis have removed all manner of things operable—tonsils teeth appendices gallbladders—but the arthritis has lingered on. That some thoughtful students are quite ready to throw the whole theory of focal infection completely overboard is but a natural reaction.

It is quite necessary then that we attempt to reappraise the place of focal infection in the pathogenesis of disease to learn as nearly as we can the facts to guide us in our practice. Nowhere have we more urgent need of guidance than in the application of the theory of focal infection to arthritis thousands of patients are still having their tonsils teeth and even appendices and gallbladders removed—all in a sincere effort to improve their arthritic condition.

Analyzing a series of 100 cases of typical rheumatoid arthritis most of which had had one or more foci of infection removed in the treatment Cecil and Angevine decided that as far as typical rheumatoid arthritis is concerned it would appear that chronic focal infection plays a comparatively unimportant role. It seemed to them that little dependence for the cure could be placed upon the eradication of such focal infection alone a fact which has become increasingly apparent to all those who attempt a critical evaluation of therapy in arthritis. Reimann and Havens arrived at essentially the same conclusion with regard to the relationship of focal infection to systemic disease in general.

It is impossible to appraise in a mathematical statistical way the extent to which the eradication of focal sepsis contributes to the cure of atrophic arthritis for no one therapeutic agent can be completely efficacious. I am convinced nevertheless that focal infection plays a definite etiologic role in atrophic arthritis. Certain clinical impressions though lacking quantitative mathematical accuracy are not to be totally discounted. Weighed as critically as possible they lead to the conclusion that focal infection plays a definite (though again not all important) role in the etiology of atrophic arthritis. And I have gained the clinical impression that the removal of causative foci of infection under given circumstances aids to a large extent both in the progress toward recovery and in the prevention of subsequent relapses.

If we assume that atrophic arthritis is probably precipitated by infection, there must be some portal of entry. In certain cases the onset of atrophic arthritis can be traced directly to an acute systemic infection—acute tonsillitis, acute coryza, or nasopharyngitis. In the majority of cases, however, such a relationship is not evident. There is good reason to believe that among these cases the most likely source of the arthritic infection is one or more of the usual chronic foci, such as infected lymphoid tissue in the tonsils and nasopharynx, infection in the nasal accessory sinuses and so on. Ample evidence, both clinical and experimental, has been presented to justify the assumption that chronic focal sepsis may play an important etiologic role in atrophic arthritis.

The author is aware that in many cases the removal of focal infection is not followed by any striking improvement, that some patients may get worse, and that others actually develop atrophic arthritis new even months or years after. However, these circumstances do not necessarily invalidate the theory of focal infection in relation to the pathogenesis of atrophic arthritis. The etiologic background of this condition results from a convergence of so many factors that striking improvement can scarcely be expected to follow the mere removal of focal infection once the disease is fully established. Removing the original focus, even if it is causative, merely eliminates a possible portal of entry for further infection, thereby aiding in conserving the general resistance, it may not influence in any direct way the disease already established in the articulations. Nor can it influence the many ramifications of the disease, noninfectious in nature, which play such a concerted role in the production of the arthritic syndrome. Though admittedly caused by a gonococcal focus of infection, gonococcal arthritis is seldom cured by treatment directed only to the original source of infection. In addition to treating the primary gonococcal focus, it is necessary to shield the affected joints from additional damage, and to employ measures which promote resolution of the inflammatory process and healing. In atrophic arthritis also the removal of a causative focus of infection is merely one step in treatment. Little progress need be expected unless, subsequent to removal of causative foci, all effects of the disease are treated adequately enough for the entire process to be controlled.

It is a well recognized clinical fact that the therapeutic value of the removal of focal infection is far greater if carried out early in the course of the disease, rather than late when there is already well established pathologic change, destruction of cartilage, and proliferation of periarticular tissue. Indeed, removal of focal infection late in the course of the disease will probably contribute little toward recovery of the much damaged joints. To conclude that focal infection plays no part in atrophic arthritis simply because patients in advanced stages of the disease do not benefit from the

removal of the infection, is like attempting to establish that syphilis is not related to tabes dorsalis because the results of antisyphilitic therapy in the latter may be disappointing

It is understandable, too, that an existing arthritis may progress after the removal of causative foci of infection. This has sometimes been true because, after removal of the presumed focus of infection, treatment was stopped or was carried out in a desultory fashion. On the other hand, many patients, though seemingly unresponsive to the eradication of focal sepsis, subsequently achieve marked improvement or complete cure when a well rounded program of treatment is instituted, that is, when the patient is given a chance to combat the many phases of his systemic disorder.

In Billings' first paper on the relationship of chronic focal infection to arthritis, in 1912, he recognized that the mere removal of causative foci of infection was not enough. Although he stressed the importance of removing existing foci, he stated it was 'just as necessary to continue a long and yet variable rest treatment with good food, restorative tonics and the various forms of individual treatment usually employed in the management of this group of diseases. No one would say that the treatment advocated is specific in that all patients are made whole and well by it, but it is believed that there is a principle involved as a cause of systemic disease which should be recognized should be sought for more frequently and when the focal infection, wheresoever it may be located, seems to be related to the systemic disease, radical measures should be instituted to remove it'. And he adds 'In those patients who, for some reason, could not or would not follow out the details of after treatment, rest, etc., improvement was not as soon obtained or not as fully secured as in those patients under command.

It is more difficult to explain the relationship of focal sepsis to atrophic arthritis when the disease begins after the removal of all apparent avenues of focal infection. In not a few patients we have obtained a history indicating that the onset of the disease followed the removal of such presumed foci.

Proof of what part focal sepsis plays in the production of the disease in these cases is not available. It is possible that in such cases the arthritis is actually a delayed manifestation of the infection. It is equally possible that bacterial invasion, and perhaps even actual pathologic change in the joints exists for months, or even years, prior to the appearance of the clinical manifestations of the disease. It may be that concealed foci of infection, not readily detected by clinical means, are the cause. The possible relationship of infection in the nasopharynx and intestinal tract must be considered. Consideration must also be given to the possibility of residual secondary foci (in joint tissues and elsewhere) which might perpetuate the

disease. The bone marrow, for example, may be one such important focus of infection. We are impressed by the fact that we were able to culture streptococci from the bone marrow in late cases of atrophic arthritis more often than from any other tissue. Other indirect evidence indicates that the bone marrow, in addition to being a possible primary site of infection in atrophic arthritis, may also serve as a niche for deposition of infectious debris, brought there by phagocytic cells operating in the joints during active stages of the disease. It is quite possible, then, that such secondary foci may perpetuate an atrophic arthritis long after the original focus of infection, accessible to surgery, has been removed.

In a previous section we have indicated the possibility of transitory bacteremias in normal individuals. Such bacteremias may conceivably induce localization of infection in joints, without producing symptoms. Latent, chronic articular infection, without symptoms, is not infrequently observed in patients who have passed through an attack of atrophic arthritis. The disease may subside to such an extent as to leave the patient entirely free of symptoms, yet permit recrudescence of the arthritis at a subsequent time when either local or general resistance is lowered. We have met cases of essentially quiescent atrophic arthritis, that is, without symptoms or objective evidence of change in the joints until the disease flares suddenly into violent activity in such articulations as are subjected to accidental trauma, as from a fall. The possibility suggests itself that in such cases the disease was clinically inactive, the pathological process was temporarily quiescent, but ready to renew activity when induced by injury.

From the available evidence one must conclude that all factors must be considered, that in relation to atrophic arthritis focal infection must neither be ignored nor considered a sole factor. One must further conclude that a well rounded and sustained course of treatment, based on a broad concept of contributing factors, is a prerequisite to cure or alleviation of the disease.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527.]

CHAPTER VII

THE VARIOUS FOCI OF INFECTION IN ARTHRITIS

The tonsils and nasopharyngeal lymphoid tissue constitute the most common foci for bacterial infection in atrophic arthritis. A history of repeated attacks of tonsillitis helps in turning attention to the tonsils as a possible site of chronic infection. This is particularly true if the patient has had quinsy or if definite chronologic relationship has been noted between attacks of sore throat or tonsillitis and the onset or exacerbations of the arthritic process. However the absence of such a history does not exclude the possibility of tonsillar infection; one may yet find contracted, scarred, partially embedded tonsils from which liquid, creamy pus may be expressed. Such findings indicate the existence of tonsillar sepsis, probably the result of repeated episodes of low grade chronic infection in which the symptoms were either so mild or so transitory as not to have been noticed by the patient.

On the other hand a history of acute attacks of sore throat may alone suggest the probability of tonsillar sepsis. Examination may disclose relatively innocuous appearing tonsils and anterior pillars only slightly reddened; pus may or may not be expressed. In such cases tonsillar sepsis may be proved only after enucleation of the tonsils.

It may be most difficult to determine what should be done about the small, firm, pale tonsils which may be largely embedded in scar tissue because of previous episodes of infection. Due to the openings of the crypts being sealed by scar tissue there may be no pus or any type of secretion on pressure. Whether the small size of such tonsils, particularly if found in older individuals, is the result of atrophy or of partial fibrosis from previous infection is not easily determined. The presence of a heavy, reddened band along the anterior pillars, extending backward to the soft palate, is said to be indirect (but not conclusive) evidence of the existence of infection in such cases. Here again the history and some demonstrable evidence of a relationship between such tonsils and the systemic disease may determine what should be done about them.

Particularly good judgment is required in evaluating the significance of tonsillar tags remaining after operations. Minute buds of lymphoid tissue

that may be seen in an otherwise clean tonsillar fossa have never impressed the author as being of importance. Indubitable evidence of infection in such lymphoid tags must be secured to justify their removal. However, larger tags, sometimes buried in scar tissue precluding any inspection of what is beneath, may be of clinical significance. This is particularly true if the history suggests that there was good reason for the tonsillectomy.

Too little attention is generally given to the condition of the lingual tonsils. Actually, they may be as important a source of systemic infection as the faucial tonsils. Again, little thought is generally given to the possibility that the nasopharyngeal lymphoid tissue (particularly in very young patients) may act as a source of infection or to the possibility that following the eradication of tonsillar or sinus infection the nasopharyngeal mucosa may act as a continuing source of infection. Nevertheless, it appears that in some cases these sources constitute major obstacles to the complete eradication of focal sepsis.

DENTAL INFECTION

Dental sepsis is another common focus of infection in atrophic arthritis, and it has received much attention. On the assumption that with the removal of the teeth the arthritis will disappear, many patients with arthritis have had not only infected teeth but even good ones removed. The dental cripples thus created may even exceed the arthritic cripples created by dental sepsis.

Obviously, frank dental infection constitutes a hazard to well being, particularly in the patient with atrophic arthritis, and, under ordinary circumstances, the removal of such teeth is thoroughly justified. There is a possibility that devitalized teeth, which through thickening of the periodontal membrane have produced a diffuse area of rarefaction about the apex, are as active sources of dental infection as teeth with localized periapical abscesses. The latter may be well encapsulated and perhaps, therefore, less capable of disseminating bacterial products.

The attitude to be assumed toward devitalized, but well filled teeth, is less clearly defined. From a bacteriologic standpoint there is evidence that many such root canals, even those seemingly perfectly treated and filled, harbor organisms of various types, particularly streptococci, of low grades of virulence. Clinically, however, it is impossible to establish any relationship between such devitalized teeth and the arthritic process. Whether they are removed or not depends on how radical or conservative one is about eradication of even suspicious sources of focal sepsis. Though in the past the author has recommended rather generally the removal of all devitalized teeth in the patient with atrophic arthritis, at present our attitude

is that such devitalized teeth may be left in with impunity. Consideration is given as to whether or not the root canals are well filled and whether there is any evidence of thickening of the peridental membrane. Clinical experience has justified the attitude that a tooth with root canals well filled and which does not reveal any reaction in the peridental membrane need not be disturbed. On the whole it seems that the importance of the relation of pulpless teeth to arthritis and other systemic affections has been exaggerated in the past.

Purrrhea and other types of gingivitis requiring as they do treatment on their own account present no particular problem as to management in the arthritic patient.

INFECTION IN THE NASAL ACCESSORY SINUSES

There is wide difference of opinion as to the etiologic importance of sinus infections in atrophic arthritis. The significance of this factor cannot be measured by statistics as to the percentages of cures or improvement after treatment of sinus disease. As we have indicated, evaluating the importance of a given focus of infection in arthritis is not a simple matter. One conclusion is clear: existing sinus infection in an arthritic is in no sense an asset.

The detection and management of existing sinus infection may be very simple or very difficult. Suppurative sinusitis producing pain and purulent discharge is easily diagnosed and its presence confirmed by transillumination, roentgenograms or sinus puncture. Whether such sinusitis is related to the arthritis or not, there is no doubt as to what is to be done. The problem of latent sinusitis in which the local symptoms are deceptively slight in degree is more difficult. The following symptoms may be significant: frequent head colds which have a tendency to chronicity, morning clearing of the throat, chronic cough. In certain localities where climatic and atmospheric conditions are poor, such symptoms may result from simple irritation without infection. However, one cannot accept such an explanation without adequate study of the sinuses by a competent, preferably conservative otolaryngologist. Examination may then reveal indirect evidence of sinusitis: purulent discharge at the ostia, congestion or edema of the nasal mucosa, swollen turbinates, and so on. Merely to refer such a patient for roentgenographic examination of the sinuses is not enough. The roentgenogram may be negative when the sinus infection is quite pronounced. The x-ray film may show opacity of the sinuses caused by thickened membrane, the end result of an ancient sinusitis long since burned out. Transillumination is equally unreliable. For this reason it is frequently necessary to employ sinus drainage either after shrinking the mucosa

(Proetz technique), or through sinus puncture and irrigation. These procedures are probably of the greatest value in diagnosing sinus infections which might otherwise escape detection. Should sinusitis be discovered, it may frequently be cleared up by conservative measures, which should by all means be tried before radical surgical procedures are employed. Allergic factors, which may constitute the basis for sinusitis and its chronicity and tendency to recurrence, must also be considered.

FOCI OF INFECTION IN THE GENITO URINARY TRACT

Although infection in the genito urinary tract may occasionally be found to be closely related to atrophic arthritis, clinical experience indicates that on the whole it is a relatively unimportant source of focal sepsis.

In addition to frank gonococcal infection, one must look for nonspecific chronic infection in the upper urinary tract, in the prostate and seminal vesicles in the male, and in the pelvis and cervix in the female. Nonspecific infections in the genital tract may be engrafted upon tissues previously damaged by gonorrheal infection.

Taylor (1937) emphasized the importance of the urologic history during the search for such infections. He pointed out that a negative urologic history will lead to fruitless search for focal infection in the urologic tract in approximately 95 per cent of cases, for in only 5 per cent of patients who had no symptoms referable to the genito urinary tract did he succeed in finding a focus of infection there. This experience, based on a study of 1,000 arthritic patients, serves as a preliminary guide to the selection of patients for more intensive urologic study.

As for the upper urinary tract, the author has found that unless there is a history directly indicative of the possibility of infection there, or unexplained pyuria, one is not justified in exploration of the ureters or kidneys in search for some concealed focus that might be related to atrophic arthritis, the distinction must be drawn between thorough investigation and purely meddlesome probing. Should such urinary tract infections exist, however, they must obviously be treated, whether a relationship to the arthritis can be established or not. Fortunately, the therapeutic means at our command today, including mandelic acid preparations and sulfamidamide, have simplified to a large extent this aspect of the problem.

Nonspecific prostatic infection, an extremely frequent condition, may exist in the absence of previous Neisserian infection. In many cases the nonspecific infection is latent and produces no far reaching effects. In others, the prostate is a most important source of infection and, when so, most pernicious and resistive to treatment. Frequently there is associated infection in the seminal vesicles, and sometimes in the posterior urethra.

(the verumontanum) as well. The diagnosis of seminal tract infection is not difficult. If there is reason for suspecting infection in the prostate or vesicles and the first examination yields a normal secretion it is wise to repeat the examination at a subsequent time when a frankly purulent discharge may be obtained. It is at times well to do a preliminary instillation of a mild silver nitrate solution into the posterior urethra and then massage the prostate and examine the secretion.

CHRONIC INFECTION IN THE GALLBLADDER AND APPENDIX

The chronically infected gallbladder is not often an important source of systemic infection. Studying 50 patients with chronic rheumatic disease, Hartung and Steinbrocker concluded that gallbladder infection plays a relatively unimportant role as a focus of infection. Statistically that is true, for the incidence of gallbladder disease is no greater in patients with arthritis than in other individuals. However the importance of an existing focus of infection in the gallbladder cannot be minimized. The author has observed a number of instances in which the existence of chronic cholecystitis generally with calculi necessitated operation because of the condition of the gallbladder itself without reference to the associated atrophic arthritis. The course of the arthritic disease in these cases was seemingly modified favorably by removal of the infected gallbladder. Particularly impressive were several instances in which acute exacerbations of chronic cholecystitis were apparently related to the onset of mild atrophic arthritides which subsided promptly following cholecystectomy. Because of these experiences the author feels that proved cholecystitis in a patient with atrophic arthritis should tip the scales in favor of operative removal of the gallbladder providing of course there are no contraindications.

Since cholecystectomy when feasible is the therapeutic indication in many cases of proved gallbladder disease incrimination of the gallbladder as a focus of infection does not materially affect the therapeutic regimen. Obviously however removal of the gallbladder on mere suspicion that it is acting as a focus of infection is never justified.

The same principles that apply to the chronically diseased gallbladder apply to the consideration of the chronically inflamed appendix as a source of focal infection.

THE INTESTINAL TRACT AS A FOCUS OF INFECTION

Anal cryptitis which may be associated with chronic perianal abscess or may exist at times without serious symptoms may act as a concealed focus of infection for atrophic arthritis as it did in at least one of our cases. It is conceivable that a chronic fistula in ano may act in the same way.

The relation of intestinal bacteria or toxins to atrophic arthritis is still debatable. Conclusive data have never been presented to prove that colonic infection plays a prominent role. Stabler and Pemberton found no difference between the intestinal bacterial flora of patients with arthritis and that of normal persons, even though the number of bacteria was greater in the patients with arthritis than in the normal controls. Nor was any significant change observed in the nature of the intestinal bacterial flora after various kinds of treatment, even when improvement was manifest. However, there are certain special cases in which such a relationship exists. Thus, in chronic ulcerative colitis arthritis occurs in nearly 5 per cent of the cases at some time during the course of that disease.

Here is proof that, at least in certain conditions of the colon infection does spread from the bowel to other distant organs. And, incidentally, it lends credence to the idea that infection from any source may enter into the causation of atrophic arthritis. It certainly suggests that the colon, seething with bacteria, normally kept in their place, may, under conditions of structural or functional disturbances of the bowel, allow the bacteria to traverse the border into the circulation. It is the author's opinion that in the management of patients with atrophic arthritis consideration of the intestine, as a possible source of influence on the disease, should not be omitted. The beneficial results that sometimes follow proper attention to the condition of the colon are striking enough to justify the assumption that some causative relationship between abnormalities in the intestine and atrophic arthritis may exist. If treatment, conditioned by such a view, produces beneficial results in some cases, the end has justified the tentative acceptance of that view.

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CHAPTER VIII

ATROPHIC ARTHRITIS

III. CLINICAL MANIFESTATIONS AND DIAGNOSIS

It is manifestly impossible to describe a course which atrophic arthritis might follow in such a manner that it will serve as a description of the disease in every patient. There are so many variations in the mode of onset in the severity and progress of the condition and in the joints involved in different cases that we may attempt to draw only a composite picture based on the observations and wide experience of the physician.

Age and Sex Incidence

Atrophic arthritis is chiefly a disease of young adults. Generally it occurs between the ages of twenty and forty, but it may set in at any age. The author has seen typical instances in a child of three and in a man of seventy-five. It attacks females three to four times as frequently as males.

The Course of the Disease

The onset of the disease may be acute. An individual seemingly well may suddenly develop pain and swelling in one or more joints which may become red, hot, and sensitive to touch. The patient is likely to show the effects of an acute systemic intoxication. He complains of general malaise and loss of appetite. The temperature may be elevated to 101° or 102°F with the pulse proportionately accelerated, and the patient may perspire freely. Digestion may be impaired and the bowels may become either constipated or loose, with the diarrhea sometimes distressing. When the onset of the disease occurs so acutely, swelling and redness of the joints may be pronounced. The ligaments and muscles about the joint become spastic and painful, limiting motion. Exudation of large amounts of synovial fluid may occur, particularly in the knees (Fig. 9). The effusion may fill the joint cavity and even distend it, further impairing motion.

Exposure to cold and dampness may precipitate this type of arthritis. The symptoms may first manifest themselves as generalized aching all over the body. Shortly after, the pain may localize in one joint, ultimately af-

fecting every articulation. It may be difficult at first to differentiate the condition from an attack of acute rheumatic fever.

Such acuteness in the onset of the disease is not common. More often

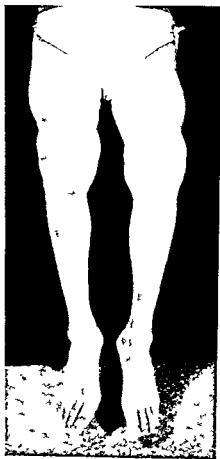


FIG. 9. Spindle shaped legs in atrophic arthritis produced by chronic synovitis of the knees with effusion: the distended joints are brought into prominence by atrophy of the muscles above and below the knees.

atrophic arthritis creeps in insidiously and may actually exist for months before the patient is seriously aware of it. For weeks he may experience fleeting but recurring pains in the muscles so evanescent that they do not constitute even a warning. There may be recurrent attacks of torticollis or lumbago which clear up completely each time. Or the patient may experience stiffness in the muscles on rising when he is not so spry as usual. After exercising he limbers up and is himself again. Later swelling and pain in one or more joints develop and for the first time the patient becomes aware of the onset of the arthritis.

Sometimes the onset is even more insidious. Although the affected joints may be painful and sore on movement, there may be little or no swelling about them and practically no constitutional manifestation severe enough



FIG 10 The thin wan haggard countenance so characteristic of the patient with active atrophic arthritis. This patient presented a subacute arthritic process of six months duration had lost nearly 26 pounds of weight and presented a brownish pigmentation of the face and of the exposed parts of the neck and hands which resembled that seen in pellagra and was in this instance probably a manifestation of vitamin B deficiency.

to arouse suspicion of what is ahead. At the outset, such an arthritic process may not appear at all serious. Confident of prompt recovery, neither patient nor physician may feel any uneasiness. Treatment is then likely to be long deferred. With absence of swelling at the joints the patient is likely to continue at work, damaging the articulations further by trauma of physical activity. Actually, such a process may become extremely stubborn and progressive, unless brought under control promptly through intensive treatment.

Even before the frank manifestations of arthritis appear, the patient may notice impairment of appetite and loss of weight. Unusual fatigue, general malaise, an inability to carry on with usual activities of work or play, perhaps a low grade fever, the origin of which cannot be traced—these are manifestations which frequently precede the appearance of fully developed atrophic arthritis. Generally, little attention is accorded these

prodromal symptoms. The patient may find that he must draw on all his reserve energy in order to carry on. For a while he may get along well. As the symptoms become more pronounced, he may be forced to retire imme-



FIG. 11. The same patient as the one pictured in Figure 10 after eight months of rest in bed and treatment of the arthritis during which time he was progressing toward recovery.

diately after work. In this way the prearthritic stage may be extended for a considerable period of time. It would be well if at such a time an attempt were made to abort the rheumatoid process which is impending.

During this prearthritic stage a wide variety of other symptoms may appear. There is frequently increased susceptibility to upper respiratory infections. There may be tachycardia and breathlessness, headache, numbness and tingling of the fingers, excessive sensitivity to cold. Sometimes fairly typical manifestations of mild Raynaud's disease appear.

Once the arthritis itself is under way additional constitutional symptoms of varying degrees of intensity generally develop. These may become aggravated as the disease progresses. In addition to loss of appetite, vague digestive symptoms may appear, particularly a tendency to bloating and gaseous indigestion. The patient appears tired and sick, the tongue is coated and

moderate grades of anemia usually develop. In time weight loss may become evident progressing in severe cases to the point of actual emaciation. The skin may become pruritic or pallid even when anemia is absent. A vel-



FIG. 12. Olecranon bursitis with effusion in a case of atrophic arthritis. Such a bursitis is the result of pressure from leaning on the elbows.

lowish to brownish pigmentation may develop during the active stages of the disease, disappearing as the patient gets well (Figs. 10, 11). Or the skin of the extremities may become atrophic and glistening, cold and clammy.

Frequently mild tachycardia exists and, in active cases, low grade fever. The blood pressure is generally low. Typical rheumatic valvular disease occurs in some cases of atrophic arthritis (probably in less than 10 per cent). Carditis is more likely if the onset of the arthritis occurred at an early age, before the third decade. Such cardiac lesions occur with greater frequency when there have been antecedent recurrent attacks of rheumatic fever. Contrary to what occurs in rheumatic fever, abnormalities are infrequently observed in the electrocardiogram of patients with atrophic arthritis.

Subcutaneous nodules, varying greatly in size, are observed in about a fourth of all typical cases. They appear at points of pressure over the extensor aspects of the arms, over the olecranon in association with effusions in the olecranon bursae, over the dorsum of the hands, the lower spine and the occiput (Fig. 12). Usually these nodules are not painful or red unless exposed to excessive pressure, in which case they may be tender and likely to remain more or less permanently.

In general, the severity of the systemic manifestations is proportional to that of the arthritis. There are notable exceptions, however, in which the

systemic disturbance is more striking than the condition in the joints that is though a generalized active arthritis is presented the outstanding clinical feature is the evidence of systemic toxicity. The clinical picture in such cases may resemble that of the more fulminating type of acute arthritis. There is the moderate fever, the extremely poor appetite, rapid loss of weight and strength and at times the distressing diarrhea depleting the body of fluids and interfering further with nutrition. Such cases substantiate the idea that infection is the most probable cause of this type of arthritis.

If the joints first involved are those of the hands as is frequently the case, the proximal phalangeal joints are those most likely to be affected. The swelling assumes a spindle shape bulging directly over the joint and tapering at both ends (Figs 13, 14). The metacarpophalangeal joints may also be affected early. There is pain, stiffness and soreness on motion, tenderness on pressure and a sensation of boggy and elasticity as if there were fluid in the joint and in the soft tissues. After a time unless precautionary measures are employed the fingers tend to assume an ulnar deviation.

The arthritis may not begin in the hands but instead begin in one of the larger joints such as the knee, elbow or ankle, spreading from there to several of the other large joints; it may remain localized to one or two joints or it may spread from one joint to another and eventually become widespread.

The small joints of the feet, either the intertarsal or the metatarsophalangeal, may be the first affected. In the feet pain is apt to predominate in the early stages and swelling may be so slight as to be altogether imperceptible. The patient frequently concludes that foot strain from fallen arches is the cause and may purchase several sets of arch supports before he is convinced that he cannot effectively rid himself of his trouble in that way. Unfortunately the physician first consulted may agree with the patient's view that the arches are actually at fault and prescribe still another pair of supports with equally disappointing results.

In typical atrophic arthritis involvement of the small joints of the hands or feet is likely to occur bilaterally and symmetrically. Not infrequently there is simultaneous involvement of the hands and feet and in addition asymmetrical involvement of other and larger joints. In the fingers and toes the proximal interphalangeal joints are most likely to be affected; the distal joints seldom.

Involvement of the wrists also frequently bilaterally may occur early in the disease. In addition to pain and soreness swelling may be very pronounced. The tendency to flexion deformity is manifest early and is facilitated by muscle spasm and improper placement of the hands if pain forces



FIG 13 Spindle shaped fingers characteristic of atrophic arthritis produced by swelling at the middle phalangeal joints

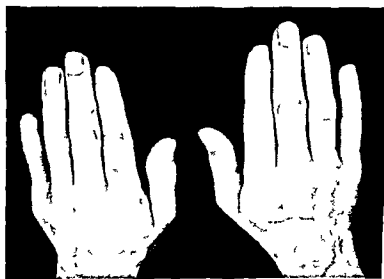


FIG 14 Disappearance of the spindle shaped deformity of the fingers with cure of the arthritis in the hands after eight months of systemic as well as local treatment of the affected joints

these joints into immobility. The capsule may become distended, permitting in addition to flexion deformity varying degrees of subluxation at the wrist. The intercarpal joints suffer severely from the damage of atrophic arthritis. It is remarkable in how short a time the carpal bones may be completely denuded of cartilage, and the wrist joints exposed to the hazard of ankylosis. Only most painstaking care and protection early in the disease can effectively limit the destruction to which the wrist joints are so subject.

The shoulder joints, though frequently affected, seldom suffer severely from destruction of cartilage, but the periarthritic involvement at the shoulders may lead to extreme restriction of motion, at first through pain and muscle spasm, and later through contracture of the capsule. In either case, the end result may be very disabling.

When there is widespread joint involvement, the cervical spine and temporomandibular joints may be affected early. The hips are least frequently affected in atrophic arthritis.

The characteristic deformities that may develop in various joints, and the factors which produce them, are of such importance that we shall accord the subject separate consideration in a subsequent section (page 183).

It is only natural for the patient to be tempted to spare his joints in order to avoid discomfort. If the joints of the legs are affected, for instance, he may take to his chair or to bed, placing the extremities in flexion, which is the most comfortable position for him. This position favors contracture of the hamstring muscles. Subsequent stretching of these muscles in attempts to straighten the legs causes still more pain, so the patient slackens in his efforts. By doing so, however, he forges the first link in the chain of events which, coupled with the inflammatory process, produces the serious deformities too often associated with atrophic arthritis. Whether the arthritis begins acutely or more insidiously, the muscles atrophy, partly because of disuse enforced by pain, partly because of the inflammatory changes in them. With the pathologic changes in the joints progressing, the wasting muscles impede motion still further, the vicious circle is established. Contracture of the muscles bringing the apposed surfaces of the degenerating cartilages and bones closer together, favors fusion in an abnormal position. The tension of the stronger sets of muscles undergoing shrinkage may induce dislocation of the bones, so that gross distortion may result.

Perhaps the most regrettable aspect of the situation is that it need not occur. Crippling deformities are not a part of the natural course of rheumatic disease; they seldom occur when adequate means for their prevention are employed at the proper time. The popular concept that arthritis and deformities are necessarily and inevitably associated is, in the author's opinion, erroneous. Deformity is no more inevitable in arthritis than is brain

abscess in mastoiditis. Both represent complications of a primary disease, complications generally resulting from neglect and only rarely from the fulminating nature of the primary disease.



FIG. 15 An arthritic derelict with flexion and adduction deformities at the hips, flexion deformities at the knees and clonus of the feet with ankylosis in these positions.

Muscle atrophy and contracture may produce a stiff joint even when changes in the cartilage and bones are still relatively slight. In such cases it may be difficult to determine clinically whether the deformity is the result of bony ankylosis or of splinting by rigid, scarred capsule and muscle. The fact remains that such deformities incapacitate the patient (Fig. 15).

Eventually the activity of the process burns itself out; pain disappears. Nevertheless, the patient remains useless for his occupation. Generally he undergoes one more stage, one rarely duplicated in extent by any other disease. He loses morale completely and understandingly enough, for in no other disease is it so subject to stress. He is weary of trying; he loses all interest in getting well.

Fortunately, atrophic arthritis does not always progress to complete invalidism. In most cases the disease is limited to only a few joints. Even if it is more widespread, the condition is apt to be more benign, with pathologic changes less severe. The damage to the joints is less profound and the general toxic effects of the disease so mild that they smoulder imperceptibly in the background. Generally the course of arthritic disease

points to nature's apparent effort to thwart the progress of the pathologic change, and it is frequently arrested before much damage has been done. Thus atrophic arthritis may burn itself out occasionally without producing serious disability, even when treatment is entirely neglected, though too often much havoc has been wrought by the time activity of the pathologic process ceases.

DIAGNOSIS

In advanced stages atrophic arthritis presents a distinctive clinical picture easily recognized. Earlier the manifestations may be so varied and confusing that the differential diagnosis may not be easy. In such cases the clinical symptomatology and the course of the disease must be analyzed minutely. Helpful supplementary data may be obtained through laboratory investigation.

The Blood Count

Various grades of anemia are generally encountered. During the acute or subacute stages of atrophic arthritis there may be a moderate leucocytosis with a relative increase in the number of polymorphonuclear leucocytes. In the more chronic phases, particularly when there is profound systemic debility, the total leucocyte count may be normal with a low normal neutrophile count. Leucopenia and a relative lymphocytosis are occasionally noted. The total white blood cell and differential counts are therefore of little aid in diagnosis or in determining the degree of activity of the arthritic process.

The Filament Nonfilament Count

A count of the proportion of filamented and nonfilamented polymorphonuclear cells is often of definite value. It may aid, first, in differentiating between hypertrophic and atrophic arthritis and second, in determining to a degree the activity of the latter.

As is well known, various types of infections tend to increase the proportion of the younger nonfilamented polymorphonuclear cells, consequently, there is a relative decrease in the number of fully ripened, distinctly filamented neutrophiles. For the purpose of such classification the polymorphonuclear leucocytes are divided into two groups: the fully segmented and the nonfilamented neutrophiles. In the first the segments of the nucleus are connected by fine filaments of chromatin material. These are the fully mature polymorphonuclear cells which normally predominate. The nonfilamented group consists of less mature polymorphonuclear cells in which the nucleus may be altogether unsegmented. If it is segmented the

segments of nuclear material are joined, not by fine strands of chromatin but by thicker bands of nuclear material. We have adopted the criteria of Cooke and Ponder that, if any band other than a fine filament of nuclear



FIG. 16 a Types of nonfilament neutrophiles b Types of filament neutrophiles

material connects the different parts of the nucleus, it is not considered divided for the purposes of such a count (Fig 16)

It has been noted that the normal nonfilament count generally constitutes 16 per cent, or less, of all leucocytes (Farley et al, Roberts). In atrophic arthritis, the proportion of nonfilamented neutrophiles generally rises above 16 per cent when there is any activity of the arthritic process, it rises considerably above the figure when that activity is pronounced. Studying 50 cases of chronic atrophic arthritis, Steinbrocker and Hartung found the nonfilament count above normal in all, the average nonfilament count being 31 per cent. We, too, have invariably found the nonfilament count to be above 16 per cent in the cases of atrophic arthritis studied from that standpoint. An increase in the proportion of nonfilamented neutrophiles assumes particular importance in early or mild cases, in which the sedimentation rate may not be accelerated.

However, it must be remembered that in nearly half of the cases of hypertrophic arthritis, the nonfilament count may also be increased beyond the normal proportion. Steinbrocker and Hartung found the average nonfilament count in their cases of hypertrophic arthritis to be 22 per cent, in contrast to the 31 per cent found in atrophic arthritis. To be sure hypertrophic (osteo) arthritis is not entirely excluded by the finding of an increased proportion of nonfilamented cells. It is possible that the increased proportion of these cells in hypertrophic arthritis is due to superimposed

points to nature's apparent effort to thwart the progress of the pathologic change and it is frequently arrested before much damage has been done. Thus atrophic arthritis may burn itself out occasionally without producing serious disability even when treatment is entirely neglected though too often much havoc has been wrought by the time activity of the pathologic process ceases.

DIAGNOSIS

In advanced stages atrophic arthritis presents a distinctive clinical picture easily recognized. Earlier the manifestations may be so varied and confusing that the differential diagnosis may not be easy. In such cases the clinical symptomatology and the course of the disease must be analyzed minutely. Helpful supplementary data may be obtained through laboratory investigation.

The Blood Count

Various grades of anemia are generally encountered. During the acute or subacute stages of atrophic arthritis there may be a moderate leucocytosis with a relative increase in the number of polymorphonuclear leucocytes. In the more chronic phases particularly when there is profound systemic debility the total leucocyte count may be normal with a low normal neutrophile count. Leucopenia and a relative lymphocytosis are occasionally noted. The total white blood cell and differential counts are therefore of little aid in diagnosis or in determining the degree of activity of the arthritic process.

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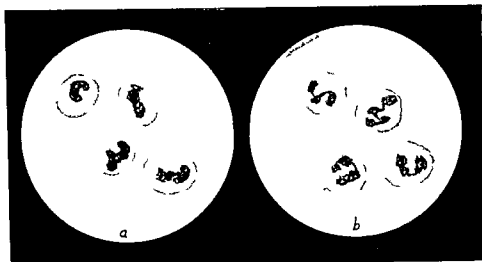


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infection in joints, or associated infection elsewhere, as in tonsils or other foci

The Sedimentation Test

If the practitioner of medicine were to limit himself to a single laboratory diagnostic measure for aid in the diagnosis of atrophic arthritis and in determining the degree of activity of the disease, he would be safest should he adopt a study of the sedimentation rate in preference to any other diagnostic laboratory procedure

In most cases of atrophic arthritis the sedimentation rate of erythrocytes is definitely and characteristically increased during the active stages of the disease. On the other hand, in hypertrophic arthritis the sedimentation rate is generally normal. It may be slightly accelerated if infection is superimposed or if the osteoarthritic process is accompanied by considerable alteration in the architecture of the joints. With these exceptions, the sedimentation test is therefore of considerable importance in the differential diagnosis of the two major types of arthritis.

We have already alluded to the fact that the increased rate of sedimentation in atrophic arthritis suggests an infectious basis for this disease. Beyond this, the explanation for this phenomenon is not entirely clear. Yardumian has shown that an increase in the fibrinogen content of the blood is one of the most important factors influencing erythrocyte sedimentation. That such an increase in the blood fibrinogen occurs in atrophic arthritis has been demonstrated by Davis, who also found a lowering of the albumin globulin ratio, another factor thought to play a part.

The process of sedimentation of red cells consists essentially of three phases. In the first the red blood cells form aggregates in rouleaux formation. In the second or settling stage the process is largely affected by pathologic changes in the plasma. The third phase begins when all of the cell aggregates have settled to the bottom of the tube and packing ensues.

It is now pretty well established that the tendency to rouleaux formation is greater in rapidly sedimenting blood. Under certain pathologic conditions something in the blood plasma apparently favors the formation of large aggregates of red blood cells. The second phase, that of actual sedimentation, depends in turn largely on the size of the aggregates formed. The third phase, that of packing of the corpuscles, is determined entirely by the number and size of the cells. Therefore the total volume of red blood corpuscles constitutes an additional, though minor factor in determining total sedimentation, the smaller the volume of red cells the greater the extent of sedimentation. Thus anemia coexisting with atrophic arthritis contributes to a slight extent in increasing the sedimentation.

Cutler has shown, however, that the effect of the cell volume becomes

operative only during the last picking phase of the one hour period arbitrarily chosen for reading the rate of sedimentation. He demonstrated (1938) that the factor of anemia influences the rapidity of blood sedimentation very little if at all. Blood sedimentation and anemia are independent phenomena and have little in common. The second phase of sedimentation that of most rapid settling is influenced only by the plasma and the size of the aggregates formed not by the size or number of cells in suspension. For this reason we have not attempted in routine practice to correct our reading of the sedimentation rate for cell volume. As Cutler (1938) pointed out attempts to correct mathematically for various grades of anemia may actually lead to erroneous computation overcorrection of the sedimentation rate and reporting of normal sedimentation when actually a moderate increase in the rate exists. Bannick also feels that from a practical standpoint routine correction of the sedimentation rate for co-existing anemia is unnecessary.

Many methods are currently employed for determining the rate of erythrocyte sedimentation (Fahraeus, Westergren, Linzenmeier, Cutler method, and various modifications). All of them yield information of value and each of them is employed by different observers. It is unfortunate that standardization of this test has not been achieved for because of the diversity of methods employed it is not always easy to compare the sedimentation rates determined by different methods.

Cutler (1926) described a method which provides graphic representation of the changing velocity of erythrocyte sedimentation at intervals during one hour. We have found this method thoroughly satisfactory and have employed it consistently.

Technique

Five tenths of a cubic centimeter of a 3 per cent solution of sodium citrate in distilled water is placed in a Cutler sedimentation tube which is graduated into tenths of a cubic centimeter and marked in millimeters.* Four and a half cubic centimeters of blood withdrawn from the vein with as little stasis as possible is added. The tube is stoppered and immediately inverted once or twice to mix the blood with the anticoagulant. The tube is then placed in a perfectly vertical position in an appropriate holder. If readings must be deferred the tube may be allowed to stand for as long as ten hours without violating the accuracy of the test. If the tube is allowed to stand for any length of time it will of course be necessary to redistribute the red cells uniformly by inverting the tube several times just before readings are begun. The position of the sedimenting column of red blood corpuscles is then read every five minutes for one hour. The observations are recorded on an appropriately ruled chart†

* These tubes are made by Arthur H. Thomas Co. Philadelphia, Pa.

† Obtainable from Charles M. Berkemeyer, Sellersville, Pa.

on which the horizontal lines represent the divisions of the sedimentation tube, and the vertical lines the intervals of time. In this way a graph is obtained which shows the position of the sedimenting column of red blood cells at any period of time during the first hour.

We report the findings in terms of millimeters of sedimentation every five minutes, but we are particularly interested in the sedimentation at the end of thirty minutes and one hour. Although the degree of settling at the end of one hour is noted, the extent of sedimentation at the end of the first half hour gives the best indication of the activity of the arthritic process, for at this time the sedimentation phenomenon is apparently entirely independent of the total cell volume. On the other hand, in rapidly settling blood, the total sedimentation at the end of one hour reflects the effect of sedimentation plus that of packing of cells. The latter is, as we have already stated, influenced by the presence of anemia.

The nature of the graph obtained depends upon the degree of settling of the cells at these periods. Essentially, there are four distinct types of graphs obtainable. From the character of the graphs they may be called (1) a horizontal line, (2) a diagonal line, (3) a diagonal curve, and (4) a vertical curve.

Employing the Cutler method, the normal sedimentation at the end of one hour does not exceed 10 mm; it averages 5 mm. The graph, plotted from observations at five minute intervals, is normally essentially a horizontal line.

When the sedimentation does not exceed 5 mm at the end of half an hour and 15 mm at the end of one hour, a diagonal line is obtained which also usually indicates the absence of active atrophic arthritis.

When the sedimentation exceeds 20 mm in one half hour, a diagonal curve is obtained. This is characteristic of active atrophic arthritis. When the activity of the arthritic process is pronounced, and a sedimentation of over 20 mm occurs within the first fifteen minutes, the resulting graph on the Cutler chart generally assumes the shape of a vertical curve (Fig. 17).

Any fundamental change in the clinical condition is generally reflected by corresponding change in the sedimentation rate. Repeated at various intervals the test may therefore serve as a reliable measuring rod of progress which may be charted on something like a quantitative basis, supplementing other reliable evidence determined by clinical means.

Return of the sedimentation rate to normal is really the most reliable objective criterion that inactivity of the process has been attained. The author has seen patients with atrophic arthritis showing marked clinical improvement (sometimes complete disappearance of pain and swelling and increased mobility of joints) but persistently retaining a rapid sedimentation rate. In former years we discounted the significance of such per-

BLOOD SEDIMENTATION TEST

Case No. 265
 Name. Mr. W. G. A.
 Diagnosis. ATROPHIC ARTERITIS

Date. SEPTEMBER 14. 1937
 Tube No. 1
 Readings by M. E.

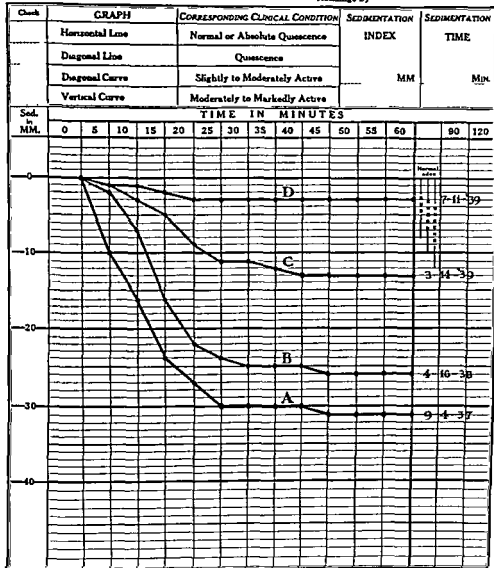


FIG 17 Graphs indicating the rate of erythrocyte sedimentation at various intervals during the course of atrophic arthritis

A Vertical curve denoting marked activity of the process before the institution of treatment (Sedimentation of 30 mm at end of one half hour 31 mm at the end of one hour)

B Diagonal curve obtained seven months later denoting less rapid sedimentation but still indicative of active disease (Sedimentation of 5 mm at end of one half hour .6 mm at end of one hour)

C Diagonal line obtained after eighteen months of treatment when the arthritic process was totally inactive and the patient clinically cured (Sedimentation of 11 mm at end of one half hour 13 mm at end of one hour)

D Horizontal line indicating normal erythrocyte sedimentation

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Case No. 265
 Name... Mr. V. G.
 Diagnosis... ATROPHIC ARTERITIS

Date... September 4, 1937
 Tube No. 1
 Readings by M. E.

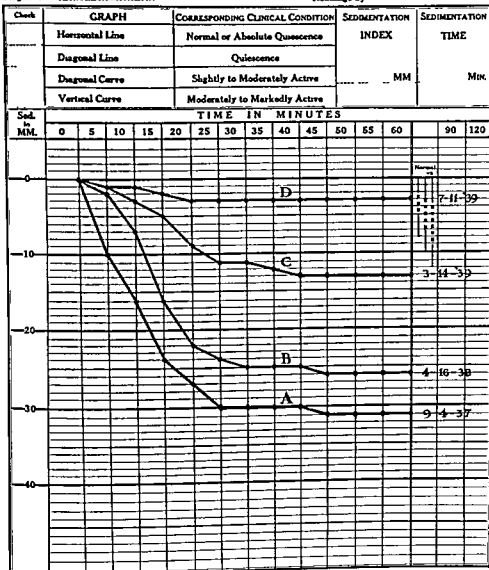


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B Diagonal curve obtained seven months later denoting less rapid sedimentation but still indicative of active disease (Sedimentation of 25 mm at end of one half hour 6 mm at end of one hour)

C Diagonal line obtained after eighteen months of treatment when the arthritic process was totally inactive and the patient clinically cured (Sedimentation of 11 mm at end of one half hour 13 mm at end of one hour)

D Horizontal line indicating normal erythrocyte sedimentation

sistently rapid rates when the clinical indications were those of improvement. In time, however, we found that such patients were subject to relapse, sometimes developing reactivation of the disease quite as severe as the initial attack. At present, therefore, we feel that clinical criteria of cure are not definitive. They should be supported by return of a normal sedimentation rate if permanency of the improvement or cure is to be assured. Holding to such criteria will also protect us against the indiscriminate and enthusiastic adoption of many therapeutic measures simply because of a temporary remission in symptoms. This may be spontaneous and not effected by the therapeutic measure employed.

Synovial Fluid

Examination of synovial fluid, aspirated from affected joints, may yield information of diagnostic value. The fluid is easily removed by tapping the joint with a large needle and syringe. In typical chronic atrophic arthritis, with symmetrical involvement of many joints and when the process is afebrile it is unnecessary to examine the joint fluid to establish the diagnosis. In acute cases, however, particularly when only one or a few joints are involved and especially if there is fever and an effusion, examination of the synovial fluid may be of crucial diagnostic importance. Routine examination includes a count of the cells and a search for bacteria, both by direct smear and culture. In atrophic arthritis the fluid is generally viscid, it may be turbid but never purulent. The cell count varies between 1,000 and 50,000 per cubic millimeter. In some acute cases the cell count may rise to 75,000 to 100,000 and even to 200,000 cells per cubic millimeter, but such counts should arouse suspicion of a suppurative type of arthritis, either gonococcal or streptococcal in origin. The polymorphonuclear cell count may range between 50 and 80 per cent, more often the former than the latter. When the proportion of polymorphonuclear cells is above 80 per cent one must again suspect septic rather than nonspecific atrophic arthritis. In atrophic arthritis bacteria are not demonstrable either in direct smears or on ordinary culture. When the possibility of gonococcal arthritis exists a complement fixation test should be performed, it is likely to be positive if the arthritis is gonococcal. A Wassermann test on the fluid need be performed only when there is suspicion of syphilitic arthritis. Chemical examination of the fluid does not, in itself, yield information of sufficient importance to justify its frequent use.

The Agglutination of Hemolytic Streptococci

Cecil has found that the serum of patients with atrophic arthritis agglutinates hemolytic streptococci in dilutions of 1 to 320, or higher, in nearly 90 per cent of well established cases of over one year's duration. Other

found that one of the earliest changes to be noted in atrophic arthritis is effusion of fluid into the periarticular structures about the joint. In the middle interphalangeal joints of the fingers the effusion generally assumes

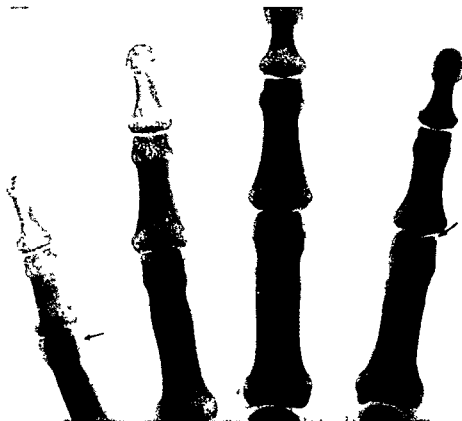


FIG. 10 Fusiform effusion early destruction of bone at the joint margin (indicated by arrows) and systemic decalcification in a patient aged thirty-one with active atrophic arthritis (From Ferguson: Roentgen Diagnosis of the Extremities and Spine, Paul B. Hoeber, Inc., 1939.)

a fusiform appearance quite characteristic for early atrophic arthritis (Figs 18, 19, 20). In the larger joints of the extremities the effusion may not present the typical fusiform appearance. When the periarticular changes are more pronounced the roentgenogram may reveal in addition to effusion thickening or swelling of all the soft tissues about the joints.

Another of the early roentgenographic manifestations in atrophic arthritis is evidence of osteoporosis caused by demineralization of the bone (Fig 21). This decalcification contrasts sharply with the appearance of the bones in hypertrophic arthritis in which the bone may be more dense than normal and may present characteristic lipping. This latter should not how

Fig. 1 Osteoporosis of the ankle caused by demineralization of bone characteristic of atrophic arthritis (Leon Ferguson Roentgen Diagnosis of the Extremities and Spine Paul B Hoeber Inc 1939)



ping may be found in atrophic arthritis in which reparative changes have occurred about areas of bone destruction (Fig. 22)
 Frequently there is associated evidence of actual loss of bony substance at the joint margins. Such bone destruction may produce irregular punched out areas similar to those in gout. In atrophic arthritis these punched out areas are generally smaller than those in gout and not so likely to be confined to the big toe. They are apt to affect many joints of the fingers or toes even symmetrically indicating thus that they are probably not gouty

ever lead one to a diagnosis of hypertrophic arthritis when there is evidence of associated demineralization of bone, atrophy, destruction of cartilage and the characteristic periarticular changes already described for such hip



FIG. 21 Osteoporosis of the ankle caused by demineralization of bone characteristic of atrophic arthritis (From Ferguson, Roentgen Diagnosis of the Extremities and Spine, Paul B. Hoeber, Inc., 1939.)

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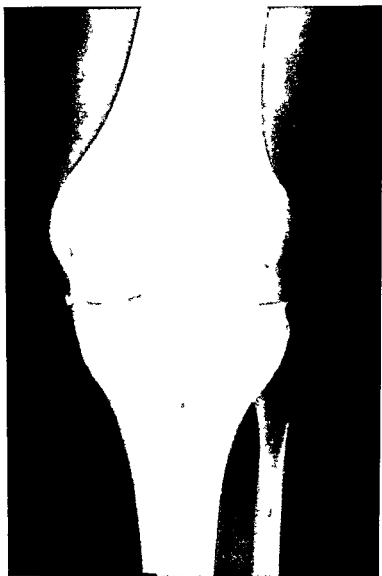


FIG. 22 Atrophic arthritis of the knee in a woman aged fifty two showing narrowing of joint space caused by destruction of the articular cartilage. There is an associated hypertrophic arthritis indicated by osteo-arthritic spurs at the articular margins of the tibia and femur.



FIG 23 Atrophic arthritis in a man aged forty, showing destruction at the third, fourth and fifth metatarsophalangeal joints, with secondary hypertrophic bony changes about areas of cartilage and bone destruction. Note also the 'punched out' areas at the affected joints, which would suggest gout, but the diagnosis of atrophic arthritis was established conclusively by biopsy.

in nature (Fig 23) The fact that such punched-out areas of bone destruction may occur in typical atrophic arthritis is not generally recognized There is a tendency to diagnose gout because the roentgenograms indicate

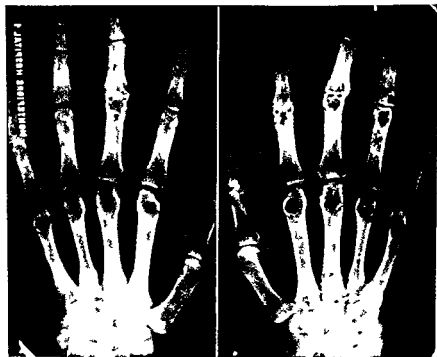


FIG 4 Atrophic arthritis of the proximal phalangeal joints of the fingers, showing discrete punched out areas resembling gout Note also the extensive destruction and matting together of the carpal bones in the wrists characteristic of advanced atrophic arthritis

these areas even when all other clinical considerations point toward a diagnosis of atrophic arthritis When in doubt because such roentgenographic changes were associated with a history of recurrent acute exacerbations of arthritis suggestive of gout we have on occasion resorted to biopsy Invariably the diagnosis of atrophic arthritis was confirmed by evidence of proliferation in the synovial tissue and lymphocytic infiltration These biopsies have convinced us that such punched out areas are produced by small tags of synovial tissue essentially small synovial villi which by proliferation and pressure apparently produce cartilage and bone necrosis of a patchy type resulting in the swiss-cheese like appearance of the joints in the roentgenogram (Fig 24)

As the cartilage becomes thinned or destroyed in the later stages of atrophic arthritis these changes are revealed in the roentgenogram by nar-



FIG 25 Atrophic arthritis of the knee in a woman aged fifty one years showing narrowed joint space from thinning of the articular cartilage and posterior subluxation of the tibia on the femur



FIG 6 Advanced atrophic arthritis of the hands showing complete destruction of the cartilages at the intercarpal metacarpophalangeal and interphalangeal joints and dislocation at some of the middle phalangeal joints

rowing of the joint space (Figs 25, 26) The degree of cartilage thinning and destruction is revealed fairly accurately by the extent of narrowing of the joint space With the cartilage entirely destroyed, the joint space disappears and leaves the ends of the bone in direct apposition Under such circumstances there is of course likelihood of ankylosis The roentgenogram however, can not be entirely relied upon for determination of complete absence of joint cartilage There are for example, instances in which the roentgenogram reveals complete disappearance of the joint space, suggesting complete destruction of the cartilage, and perhaps ankylosis, yet clinical examination may reveal a considerable (perhaps even a full) range of normal mobility Such joints may retain a film of cartilage too thin to be revealed in the roentgenogram but sufficiently thick to prevent ankylosis This may eventually be proved by the fact that with cure of the arthritis the joint retains full motion This brings out the importance of correlating the findings of the clinical examination with those in the roentgenogram in order to evaluate correctly the character and extent of the pathologic change

Naturally various characteristics mentioned may be noted in different joints of the same patient affected with atrophic arthritis By routinely studying roentgenograms of the knees, feet, and hands, it is likely that one or more of the typical changes will be discovered

THE VARIABLE COURSE OF ATROPHIC ARTHRITIS

Atrophic arthritis may pursue an erratic course Spontaneous remissions of varying degrees and for varying periods of time are essentially characteristic Exacerbations of the disease also occur, and may do so despite well directed treatment There is probably nothing that taxes the patient's courage so much as the appearance of an exacerbation, with increase in pain and swelling in the joints at a time when he is seemingly progressing toward recovery Numerous influences may affect the patient adversely, bringing sudden transitory storms when the condition of the joints seems to be undergoing resolution Frequently the factor responsible for the temporary relapse is evident, occasionally it is not However, an attempt should always be made to find the cause of these storms when they occur, for it may frequently be detected and, by proper care, eliminated

We have already indicated that one of the factors that modifies to a large extent the course of the disease is the fundamental constitutional makeup of the patient Certain individuals are inherently endowed with more, or less resistance to arthritis than others The economic status of the patient is another factor which modifies the course of this condition The reasons are obvious A patient who cannot afford the time or the money

for a necessary rest cure, or who is exposed to physical and nervous stress and anxiety, is not likely to do so well as a patient less handicapped

Intercurrent infections, such as a 'cold' or 'grippe,' may cause an exacerbation of symptoms. There may be only excessive stiffness and soreness in the muscles, or the pain and swelling of the joints may actually increase.

The ability of some arthritic patients to prophesy impending changes in the weather has been known for centuries. Many influences other than barometric pressure have been suspected as possible causes for this reaction. Among these are humidity, temperature variations, the presence or nearness of storms, currents of wind, and atmospheric electricity.

Some patients complain that certain dietary indiscretions (excesses in meats, sweets, or certain fruits, overeating) precede temporary exacerbations of the disease. The basis for blaming such factors is not at all clear; it seems more likely that they are incidental, and not related.

Women may complain of cyclic monthly exacerbations of arthritis coincident with the period of menstruation. There is no clear explanation for the fact that there may be an increased amount of pain and swelling of the joints either just preceding or immediately after menstruation.

The course of atrophic arthritis may be further modified by concomitant affections of various sorts. The author has seen cases of atrophic arthritis associated with active (though asymptomatic) syphilis. The arthritis was not primarily on a syphilitic basis, but treatment of the syphilis exerted a decidedly beneficial effect on the course of the arthritis. Equally striking is the effect on atrophic arthritis of cure of existing psoriasis in those patients in whom these two conditions coexist. When pernicious anemia is associated with atrophic arthritis, satisfactory control of the anemia may be followed by improvement in the rheumatic condition. Oddly enough, observations are on record in which the contrary holds true, that is, exacerbations of atrophic arthritis occurring coincidentally with return of a normal blood picture.

The most important factor modifying the course of this disease is the general constitutional state of the patient. The maintenance of a high degree of general resistance and well being is indispensable both for attaining cure and for maintenance of it. Many times do we encounter patients, entirely recovered from a rheumatic affection, slipping back into a relapse when, for any reason, the general body resistance is lowered.

This brings up the question of the meaning of "cure" as referred to atrophic arthritis. Is such a patient entirely cured if he is altogether free of symptoms, even to the presentation of a normal sedimentation rate and blood picture? Or is it necessary to speak of such cases as "arrested" or "quiescent" arthritis, as we speak of "arrested" and "quiescent" tubercu-

losis? We are reminded of those many instances encountered at post mortem examinations in which thoroughly healed tuberculosis in the parenchyma of the lungs was associated with active or "quiescent" miliary tubercles in the hilum lymph nodes. Here were instances of *clinically* inactive tuberculosis but *pathologically* one could not speak of the process as anything but active. We can suspect that a similar state of affairs holds true in atrophic arthritis. Though clinically the disease may be inactive, frankly active pathologic foci may lurk in the background ready to renew activity when circumstances are propitious. Like the tuberculous infection, the arthritic process is kept in check so long as general body resistance is high; it flares into renewed activity when for any reason the systemic resistance is lowered. Although many patients may attain a clinical cure, there is perhaps no such thing as a cure of arthritis pathologically. We are more accurate in referring to such cases as "quiescent" or "arrested" arthritis. Such an attitude is particularly wise because it connotes the necessity for continued observation of the patient at intervals throughout his life; it dictates the necessity for sheltering him from even the slightest excesses in order that permanency of the clinical cure might be insured.

Not every disease has a deleterious influence on arthritis, some are actually helpful. Hench and others have discussed the ameliorating effect of jaundice on chronic atrophic arthritis. The effect of intercurrent jaundice may be nothing short of dramatic. Thus the appearance of jaundice of significant degree may, under favorable circumstances, either wholly inactivate the arthritic process or lessen its severity, or at least induce analgesia of some degree. The remissions frequently last throughout the duration of the jaundice, sometimes long after and when symptoms recur, they may be much milder than those previous to the onset of the jaundice. The possible application of this principle in the treatment of arthritis will be discussed later (page 153).

Some patients with atrophic arthritis experience a similar ameliorating effect from pregnancy. Hench has contributed an interesting clinical analysis of the effects of pregnancy in atrophic arthritis. He pointed out that remissions generally appear at the end of the first month of pregnancy and usually last throughout the period of gestation, frequently continuing for a month or two afterward. The completeness of such remissions, the obvious relationship between them and the pregnancy, the consistency of the phenomenon to the extent that relief from atrophic arthritis may occur every time a given patient is pregnant, all indicate the probability that some systemic or hormonal change characteristic of the state of pregnancy is responsible.

Rawls has reported marked or complete remission of symptoms coinci-

administration of cinchophen. Relief was generally temporary, but in some instances persisted for weeks after the disappearance of urticaria. Significantly, other patients did not obtain such striking relief from urticaria developed during treatment with cinchophen. The interpretation of these observations is not clear. Whether the amelioration of the arthritic symptoms was the result of the cinchophen per se, whether it was the effect of some chemical change resulting from the toxic influence of the drug, the urticaria, or some other factor, cannot be stated. All these observations are important, however, since they point to the fact that many variations in the physiologic or chemical milieu of the patient with arthritis may induce amelioration of the process. They indicate, furthermore, the necessity for careful study of these phenomena with the idea of determining, if possible, just what chemical or physiological changes are responsible. Obviously, any light that might be shed by such investigations would advance therapeutic possibilities.

DIFFERENTIAL DIAGNOSIS

Initial attacks of acute rheumatic fever and of acute atrophic arthritis may, at the outset, be practically indistinguishable. With continued observation the diagnosis may become clear. Rheumatic fever, rather than atrophic arthritis, is suggested when the joint pains are severe with relatively little swelling or effusion, and when there is high fever, sweating and toxemia, with salicylates affording marked relief. If evidence of endocarditis or of abnormalities in the electrocardiogram should appear during the course of the acute arthritis, rheumatic fever is again the more probable diagnosis. In this condition there is also eventually complete disappearance of pain and equally complete resolution of the inflammatory process in the joints. In atrophic arthritis some residual changes practically always persist, even after the first attack.

The "specific" infectious arthritides must be excluded by specific bacteriologic, serologic, or immunologic tests. Septic forms of arthritis may be distinguished from ordinary atrophic arthritis by examination of the synovial fluid. In the former, there is generally a high total cell count, the proportion of polymorphonuclear leucocytes exceeds 75 per cent, and, frequently, the organism may be detected in direct smears or recovered in culture.

When gonococcal arthritis affects many joints, as it may do, it may be clinically indistinguishable from acute atrophic arthritis. To establish a diagnosis it should be ascertained whether there is any chronologic relationship between the onset of the arthritis and a recent bout of gonorrhea in the genital tract. A history of an old gonorrheal infection does not con-

stitute adequate grounds for a diagnosis of gonococcal arthritis unless the specific organism is recovered either from the genital tract or from the affected joints. Recovery of the gonococcus establishes the diagnosis. The gonococcus complement fixation test on the blood or synovial fluid is positive in most cases of active gonococcal arthritis, but, by itself, this test is not absolutely diagnostic for it is occasionally positive in acute cases of atrophic arthritis.

The diagnosis of tuberculous arthritis must be considered particularly when only one or a few joints are affected, especially in children. The roentgenogram may indicate tuberculosis but a diagnosis should be made before roentgenographic changes appear. Examination of the joint fluid may yield the clue to the possible tuberculous nature of the arthritis, and the diagnosis may then be confirmed by the results of guinea pig inoculation. Occasionally biopsy may be required to establish the diagnosis.

When repeated acute attacks of arthritis suggest the possibility of gout, determination of the uric acid concentration in the blood is essential. The presence of tophi or of typical roentgenographic evidence of gout clinches the diagnosis. The urine is usually normal in cases of atrophic arthritis, while in long standing gout it is more likely to show the characteristics of mild nephritis.

Hypercalcemia is practically never observed in atrophic or hypertrophic arthritis. Should it be found, one must consider the joint involvement as part of a systemic disease such as hyperparathyroidism.

This brief discussion of the many clinical and laboratory features that may be used in arriving at a diagnosis may create the impression that each bit of data is presented as definitive. This is far from true, for, while these data are without doubt useful, their fullest value in actual practice is dependent upon recognition of the many qualifications and conditions that enter into their interpretation. In a disease with so many ramifications and endless variations, any reliable estimate with regard to diagnosis or progress of the case can be obtained only through most precise evaluation of all the data, clinical and laboratory.

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CHAPTER IX

ATROPHIC ARTHRITIS OF THE SPINE

SYNONYMS *Rheumatoid arthritis of the spine, Marie Strumpell disease, spondylitis ankylopoietica, spondylose rhizomelique, atrophic spondylitis, rheumatoid spondylitis, infectious spondylitis Bechterew's disease*

One of the most common variants of ordinary rheumatoid arthritis is atrophic arthritis of the spine. It occurs chiefly as a primary involvement of the spine, although involvement of the peripheral joints may coexist in some cases.

Atrophic arthritis of the spine occurs more frequently than has been assumed. Tyson reported that in the Arthritis Clinic of the Presbyterian Hospital in New York one case of this condition is encountered to about every thirteen cases of atrophic arthritis. Like the latter, the condition under discussion is primarily a disease of adolescents and young adults, although it may occur at almost any age. Unlike ordinary atrophic arthritis, however, this type of spondylitis is predominantly a disease of males, occurring four to six times more frequently among men than women. The reason for this peculiar sex distribution is not apparent. It may be the result of greater inherent susceptibility of the spine, or it may be that trauma plays an important predisposing part in localization of the disease. By and large, the occupations of men are apt to be more strenuous, thus more prone to expose the spine to trauma. These facts may explain the greater frequency of the disease in males.

There has been considerable speculation as to the etiology of this type of spondylitis. The condition has been attributed by some authors to gonorrheal infection. This idea is no more than a supposition, however. Gonococcal infection has been found to occur no more frequently with atrophic arthritis of the spine than with many other conditions. On the other hand, considerable circumstantial evidence points to rheumatoid spondylitis as more closely related to ordinary atrophic arthritis. In the first place, the familial tendency to this type of spondylitis seems to follow a pattern much like that seen in atrophic arthritis. Not infrequently there is a history of antecedent rheumatic fever. A certain proportion of patients with

atrophic arthritis of the spine develop typical rheumatic valvular disease. The pathologic changes in the spinal articulations resemble those of atrophic arthritis elsewhere. Most important of all is the fact that rheumatoid spondylitis may be merely a part of generalized atrophic arthritis.

The early pathologic changes consist essentially of synovitis and peri-arthritis of the small intervertebral joints. Gilbert Scott of London has shown that in over 90 per cent of the cases of Marie Strumpell spondylitis there is an associated atrophic arthritis of the sacro-iliac joints. It is contended by some authors that such sacro-iliac arthritis is invariably the seat of origin of the spondylitis, the condition spreading from there to other regions of the spine. When the thoracic spine is affected peri-arthritis of the costovertebral (as well as of the intervertebral) joints develops early. In addition to the earliest peri-arthritic changes there develop in succession spasm of the muscles about the spine, osteoporosis of the bodies of the vertebrae, and thinning of the cartilage of the vertebral articular facets. In its later stages the pathologic process is characterized by calcification of the paravertebral ligaments, particularly of the anterior and lateral longitudinal ligaments, as well as of the ligamenta flava. Finally, the intervertebral and costovertebral articulations may become ankylosed and the capsules of these joints, as well as the spinal ligaments, may become infiltrated with calcium. Calcification of the lateral longitudinal ligaments and of the lateral borders of the intervertebral disks produces the characteristic bamboo rod appearance of the vertebrae as seen in the roentgenogram (Fig. 27). The type of calcification which occurs in this disease is quite different from that which produces the irregular exostoses so characteristic of hypertrophic (osteo-) arthritis. As a rule the process extends throughout the greater part of the spine, although it may be limited to certain segmental areas.

As has already been stated, atrophic arthritis affecting the joints of the extremities may be associated. When joints other than those of the spine are concomitantly involved, the hips and shoulders are most likely to be affected. It is because of this tendency to involvement of the pelvic and shoulder girdles that Marie called the disease 'spondylose rhizomelique'. When the smaller peripheral joints are involved, as is occasionally so, they present the typical manifestations of atrophic arthritis.

Sometimes the condition begins acutely with fever, severe pain, marked weight loss, and marked systemic debility. In such cases the disease may run a fulminating course, leading in a short time to marked destruction of the intervertebral joints, extreme muscle atrophy, and ankylosis of the spine. More frequently, however, the disease begins insidiously. The earliest symptoms then consist of vague pains and stiffness in the back, particularly on motion, easy fatigability, perhaps loss of weight, and other manifestations commonly associated with atrophic arthritis. Referred pain from

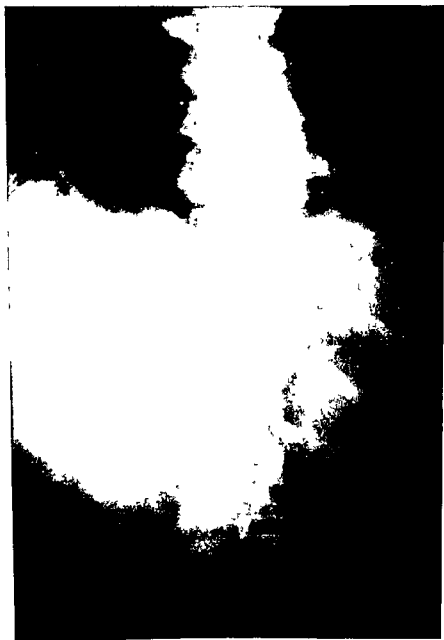


FIG 27 'Bamboo rod' appearance of spine, produced by calcification of both lateral longitudinal ligaments, in advanced atrophic arthritis of the spine (Marie Strumpell spondylitis)

irritation of the spinal nerve roots may first bring the patient to the physician. When the thoracic spine is affected pain in the back with referred pains around the chest on breathing may suggest pleurisy. Pain may be



FIG. 28 Atrophic arthritis of the spine (Marie Strumpell spondylitis) showing a rigid forward bowing at the dorsal spine. This deformity is not amenable to correction because calcification of the spinal ligaments has already developed.

referred from the lower dorsal roots across the loin or abdomen erroneously suggesting intra-abdominal visceral disease. The referred pains may radiate to the shoulders or hips even when these joints are not affected although as we have stated they are occasionally concomitants of this type of spinal arthritis. One of the early manifestations may be severe sciatic pain from involvement of either the sacro-iliac or lower lumbar articulations.

The earliest objective signs are evidence of rigidity of the spine from muscle spasm with resulting limitation of motion particularly loss of normal extension of the spine and varying degrees of modification of the normal spinal curves. When the dorsal spine is affected there is generally

increasing limitation of motion of the ribs, sometimes complete fixation of the chest, resulting at first from muscle spasm and periartitis and later from actual ankylosis. Finally, unless precautions have been taken early enough to prevent it, a forward bowing of the spine may develop, with fixation in that position (Fig 28). Thoracic respiration is entirely lost, the chest becomes flattened, with its anteroposterior diameter reduced, and symptoms of respiratory embarrassment, caused by loss of thoracic function, supervene. In neglected cases, with rigid kyphosis of the dorsal spine and complete fixation of the chest, the respiratory symptoms may assume serious proportions.

Involvement of the cervical spine leads, of course, to stiffness and, later, to ankylosis, with fixation of the head either in the erect or a markedly flexed position, depending on the posture that was maintained during the active phase of the disease. When the lumbar spine is affected and there is associated involvement of the hips, a characteristic slow, shuffling gait develops which, associated with dorsal and cervical kyphosis, presents a clinical picture that may be recognized at a glance.

It is obvious, of course, that the diagnosis should antedate any such development. During the earliest stages, when treatment is most effective, the diagnosis may have to be based solely on the early clinical manifestations, for roentgenographic changes do not appear until late. There are generally few, if any, roentgenographic changes during the early, active stages of the disease. The earliest changes to be noted in the roentgenogram are evidence of generalized osteoporosis of the spine, haziness at the intervertebral joints and, in most cases, narrowing or complete obliteration of the joint space at the sacroiliac articulations. Roentgenographic evidence of atrophic arthritis of the sacroiliac joints associated with diffuse osteoporosis of the spine should suggest the probability of Marie Strumpell disease. As the condition progresses, the roentgenogram reveals increased osteoporosis, as well as increased density of the paraspinal ligaments, followed later by calcification of these ligaments as well as of the intervertebral and costovertebral joints (Fig 29). But symptoms of atrophic arthritis of the spine may exist for years before x-ray evidence appears. If the clinician waited for roentgenographic evidence before making the diagnosis, atrophic arthritis of the spine would, in most cases, be undiagnosed for anywhere from three to six years. Meanwhile the most valuable time for effective therapy would be lost.

Other laboratory studies may be too equivocal. As in atrophic arthritis of the extremities, the blood count is generally of little aid in diagnosis except in acute fulminating cases in which leucocytosis may occur. A few cases show positive agglutination of the serum with hemolytic streptococci. The sedimentation rate may be accelerated in the most active cases.

I have found however that when the onset of the disease is insidious and slowly progressive the sedimentation rate may be perfectly normal even during active stages of the disease. This is in contradistinction to what



FIG. 9 Atrophic arthritis of the cervical spine (Marie Strumpell spondylitis) in a woman twenty five years of age showing bridging between vertebral bodies resulting from calcification of anterior longitudinal ligament

occurs in other types of atrophic arthritis. The sedimentation rate is more likely to be accelerated if in association with the spinal arthritis there is involvement of peripheral joints.

If ankylosis of the spine develops its shape is determined by the position the patient assumed during the active phase of the process. If he has been ambulatory with the spine unprotected cervical and dorsal kyphosis are most likely to develop. If the patient is forced to bed during the active stage of the disease the resulting ankylosis is more likely to be straight, the typical poker spine.

TREATMENT

Owing to the proclivity for progression and the development of serious deformity if neglected therapy should be instituted at the earliest time

Although the systemic disturbance induced by this disease is rarely as striking as in generalized atrophic arthritis, treatment with a view to improving the patient's general physical condition and increasing his resistance should be carried out as in ordinary atrophic arthritis. Evident foci of infection should be removed at the appropriate time.

Rest in bed, local application of heat, and prevention of spinal deformity by correct posture and corrective postural exercises, are most important during the active stages of the disease. If ankylosis appears inevitable despite such treatment, support of the dorsal spine in extension by means of plaster shells or jackets will leave the chest in the desired position of inspiration. These aspects of therapy are discussed in Chapters XIV and XV. Deformity caused by ankylosis is, in this condition, not amenable to correction. When the shoulders or hips are also affected, measures to prevent deformity and ankylosis in these joints must also be attempted. Unfortunately, the hips, when seriously affected, practically always fuse, regardless of the effort to prevent it.

This type of spondylitis is not infrequent. It is admittedly a most stubborn form of arthritic disease, affecting men at the most useful periods in their lives. Everything should be done to thwart its progression and the alarming sequelae. This may be accomplished more frequently than we have in the past supposed. If a patient with Marie Strumpell spondylitis becomes bent over and fixed in that uncomfortable position, it means that during the active stages of the disease proper measures for the prevention of this deformity were not instituted. The use of plaster jackets or a Taylor brace, by which the spine may be maintained in the desired position of normal extension indefinitely, not only prevents deformity, but, by relieving muscle spasm, affords relief from pain. Possibly with proper fixation of the spine and resolution of associated spasm of the adductor muscles of the legs, protection against serious damage to the hips may also be afforded.

Is such early, persistent fixation by a brace or plaster jacket likely to cost the patient freedom of mobility in his spine? Extended experience has made it strikingly evident that early fixation of the spine for long periods of time prevents excessive destruction of the intervertebral articulations and is conducive to earlier arrest of activity of the process, with less calcification and bony ankylosis. Some patients with spines quite rigid and immobile before immobilization may, in fact, after long periods of fixation, secure restitution of a considerable degree of motion. This is no doubt effected by resolution of muscle spasm and by limiting progression of the periarthritis, when the effect of trauma is removed from that of inflammation. Moreover, if ankylosis should develop during the period of fixation of the spine, as may inevitably occur in certain cases, it will occur in the most favorable functional position. Incidentally, such fixation will, of course, afford com-

plete relief from pain. In any event indications point to the fact that ankylosis developing during appropriate immobilization of the spine results not from the fixation but rather in spite of it.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527.]

CHAPTER X

STILL'S DISEASE

Still's disease is essentially atrophic arthritis as seen in children. It generally begins between the third and tenth year, although it may set in before the age of one, and sometimes appears in fairly typical form in adolescents. Still's original description in 1897 emphasized the association (with the arthritis) of enlarged lymph glands, a palpable spleen, and a tendency to chronic pericarditis. As previously stated, these unusual clinical features gave rise to the thought that this form of childhood rheumatism might be distinct from ordinary atrophic arthritis of adults, but it is now regarded merely as a variation. Lymphadenopathy, liver and spleen enlargement and anemia are probably merely peculiar pathologic reactions conditioned by the age of the patient. The marked lymphatic hyperplasia characteristic of Still's disease is probably merely an expression of the rather usual active lymphocytic response so readily produced by various types of chronic infection in children, for the arthritis is, in every other respect, identical with that which occurs in adults. Moreover, Feltz has actually described a similar syndrome in adults, that is, chronic arthritis associated with an enlarged liver and spleen, and a tendency to leucopenia.

Although from a theoretical standpoint it may be important to distinguish this peculiar type of pathologic reaction, from the standpoint of the clinician it is better to accept the idea that Still's disease is merely a modified form of ordinary atrophic arthritis.

In Still's disease the pathologic changes in the joints and other tissues are essentially the same as those in atrophic arthritis. Subcutaneous nodules are frequently associated, as is also low grade pericarditis. The lymphatic tissues reveal evidence of a subacute or chronic inflammatory process, with proliferation and infiltration of lymphocytes and varying degrees of fibrosis, but nothing that is otherwise specific. There is moderate leucocytosis in the early, most active stages of the disease, with a relative increase in the polymorphonuclear leucocytes. With increasing chronicity there develops leucopenia, with relative lymphocytosis. The sedimentation rate is invariably accelerated during the active stages of the disease.

It seems probable that the same causes that operate to produce atrophic arthritis of the adult are concerned in the etiology of Still's disease. Focal

infection is regarded as having an important relationship to this disease. There are isolated reports of dramatic improvement shortly after the eradication of infective foci in early stages of this condition.

Although the onset of the disease is usually insidious, like that of the ordinary type of chronic atrophic arthritis it may develop very acutely and follow a fulminating course. Though in general Still's disease follows a course similar to that of atrophic arthritis of the adult, quite commonly its systemic manifestations assume more striking proportions. The per articular joint swelling too may be very marked assuming exceptional proportions as it stands out in striking relief against the background of wasted atrophic muscles. The glandular enlargement, which may affect any group of lymph nodes and the spleen may assume very marked proportions or may be so slight as to be barely perceptible. The severity of the adenitis does not always parallel the severity of the arthritis. Typical rheumatic endocarditis develops in a small proportion of cases, but not nearly as often as in rheumatic fever. Pericarditis is a not infrequent concomitant. Usually not evident during life it is discovered only at autopsy. Unless adequate preventive steps are taken the changes in the joints are apt to be progressive with deformity and ankylosis developing quickly.

There is a tendency for the persistence of infantile proportions of the limbs and for the development of dwarfism in some of these children. This has led to discussion of a possible endocrine basis for the disease, but neither clinical nor pathologic indications exist of any primary endocrine abnormality in relation to this condition. With the occurrence of such a systemic disease in children at an age when the ductless glands are actively concerned with stimulating growth and development, it is conceivable, of course, that some impairment of endocrine function might occur. Furthermore, such a condition is accompanied by profound nutritional disturbances which may contribute to disturbance of glandular function. Such endocrine disturbances as might occur may, then, be but a reflection of the markedly altered, general physiologic status of the patient. It appears that the most important factor conducive to dwarfism and maldevelopment of the extremities is probably a local disturbance at the epiphyseal growth centers of the limbs, adjacent to affected joints.

The roentgenographic changes observed in Still's disease are in all respects similar to those seen in atrophic arthritis of adults. Some writers have emphasized the tendency to the development of osteo-arthritic changes about areas of cartilage and bone destruction. Such changes are not unusual, however, in advanced atrophic arthritis.

TREATMENT

The treatment of Still's disease is not essentially different from that of atrophic arthritis in the adult. Details will, therefore, not be discussed separately.

There are, however, certain special considerations that deserve mention. For example, some difficulty is encountered in securing active co-operation from these young patients. Their extreme sensitiveness to pain may cause them to spare the joints to such an extent as to invite early contracture, deformity, and ankylosis, and the physician must be prepared against such eventualities and anticipate them by adequate splinting, physiotherapy, and passive and active exercises. These measures will be described in subsequent sections (page 167) but one can hardly overemphasize this aspect of therapy for there is no more pathetic sight than that presented by a child with Still's disease, inadequately or improperly treated.

The nutritional problem presented by atrophic arthritis of children is another one that deserves special consideration. These patients are extremely liable to develop marked weight loss, disturbances of nutritional balance, avitaminoses, and deficiencies in mineral metabolism. These must be guarded against, the diet must include an adequate provision of protein for growth and must be rich in vitamins and minerals. Vitamin supplements are frequently necessary. The provision of optimum nutrition, so important in all cases of arthritis, is especially important in the treatment of Still's disease. Still's disease may remain active for from three to five years. If adequate treatment is carried out throughout this time, the chances are very good for complete cure in some cases. The outlook for recovery depends on adequacy of treatment, how early treatment is instituted, and how conscientiously and with what perseverance it is carried out. Colver, in a follow-up study of 49 cases of atrophic arthritis in children, was impressed with the number that recovered practically completely. He indicated that in those cases in which complete recovery ensued, the disease became quiescent after several years. Still's disease has a relatively high mortality rate. Most of the deaths occur in children who develop the most acute, fulminating form of the disease during infancy and early childhood, before the age of five. Many of the fatalities are the result of either intercurrent infection or carditis, complications to which these children are subject.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 52-]

CHAPTER XI

PSORIATIC ARTHRITIS

SYNONYMS *Arthropathia psoriatica, psoriasis arthropathica, psoriasis arthritica*

Psoriatic arthritis is identical with ordinary atrophic arthritis in all respects, but is associated with severe or inadequately treated psoriasis, and usually exhibits a correlation between the activity and severity of the arthritic and dermal manifestations. Frequently the psoriasis precedes the arthritis, but it may appear simultaneously with or even after the development of the latter. Once the relationship is established, the striking feature is the simultaneous occurrence of exacerbations and remissions in both conditions, although this rule is not invariable. In addition to the usual arthritic manifestations, there is a tendency toward involvement of the terminal phalangeal joints in the fingers and toes, and coexistent psoriasis, with pitting, hyperkeratosis, and distortion of the nails adjacent to affected joints.

The pathologic changes in the joints in psoriatic arthritis are identical with those of ordinary atrophic arthritis. The findings on laboratory examination and the roentgenographic changes are also indistinguishable from those of ordinary atrophic arthritis of an equal grade.

Studying 26 cases in which psoriasis and atrophic arthritis were associated, Dawson and Tyson concluded that this association is more than a chance phenomenon and that the two diseases are somehow intimately related. Their observations, as well as those of others, indicate the probability that the psoriasis and atrophic arthritis are based on some etiologic factor common to both, but precisely what the common denominator may be, remains a mystery. Recognizing the possibility of such a common etiologic factor for these two conditions suggests that any light shed on the etiology of either of these processes may reflect the etiologic mechanism of the other.

TREATMENT

The arthritis responds to measures effective in ordinary atrophic arthritis. A detailed discussion of these follows (page 129). Treatment of the

psoriasis is as essential as treatment of the arthritic condition if maximum benefit is to be attained. The treatment regimen for psoriasis outlined by Goeckerman based on the combined use of crude coal tar ointment and ultra violet therapy appears to be the method of choice. X ray and other forms of treatment are sometimes indicated and useful.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527]

CHAPTER XII

FOCAL ARTHRITIS

There is another variant of rheumatoid arthritis which some writers have preferred to call focal arthritis or chronic infective polyarthritis. It has been stated that this differs from typical atrophic arthritis in certain respects: that in focal arthritis the etiologic relationship between infectious foci and the disease is more obviously manifest, that patients with focal arthritis do not generally present the typical asthenic body habitus of the patient with atrophic arthritis nor striking evidence of familial predisposition to the disease and that in focal arthritis there are few, if any, of those constitutional manifestations typically associated with atrophic arthritis. It is also pointed out that focal arthritis is characteristically a disease of the larger joints, that it is less likely to involve the small peripheral joints of the extremities and, that the arthritic process is asymmetrically distributed and is not bilateral and symmetrical as in atrophic arthritis.

Though the clinical picture differs in some respects, it appears unlikely that focal arthritis differs essentially from the more typical atrophic arthritis, probably it is merely a variant. The characteristic behavior of focal arthritis is dependent, perhaps primarily, upon a peculiar susceptibility of larger joints to the disease upon an inherent constitutional resistance of the patient, and perhaps also on the severity of the infection. It would seem, then, that in focal arthritis the process remains localized in one or a few large joints because of an inherently lesser susceptibility to widespread arthritis and because of a relatively better resistance to the infection at the time that it occurs. That both atrophic and focal arthritis are probably alike fundamentally is indicated first by the fact that pathologically both of these forms of arthritis are essentially identical. The author has found no evidence of active focal infection to be more striking or more frequent in focal arthritis than in atrophic arthritis. The findings on clinical laboratory examination as well as the changes in the roentgenogram are essentially the same in both conditions. The most important indication of the unitary etiologic basis of these two types of arthritis is the fact that a condition starting as focal arthritis may progress into most typical atrophic arthritis, with symmetrical involvement of small peripheral joints, development of constitutional debility and so on.

TREATMENT

Because we believe focal arthritis to be essentially a form of atrophic arthritis our treatment is essentially the same for both of these conditions and will be described later (page 129). Focal infection, wherever found, should be removed early, protection of affected joints is carried out as in atrophic arthritis, and all constitutional factors entering into the process are treated as indicated. Since the joint infection is apt to be more localized, and the constitutional manifestations of the disease less severe, the results of treatment of focal arthritis are, as a rule, more satisfactory and more easily obtained than in the more typical atrophic arthritis.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527.]

CHAPTER XIII

THE TREATMENT OF ATROPHIC ARTHRITIS

The treatment of atrophic arthritis presents a challenge not only to the knowledge of the physician, but also to his resourcefulness in the art of its application. We have no specific upon which we may rely, no specific vaccine, diet, drug or physiotherapeutic agent, which, alone, will effect a cure. However, we have no reason for undue discouragement, for few other chronic diseases with such menacing potentialities are so greatly ameliorated or so satisfactorily arrested by treatment applied intelligently, adequately, and sufficiently early.

We too often meet arthritic patients with crippling and disabling deformities which might have been avoided had well directed treatment been administered early enough. At times the patient himself is responsible for his plight by having turned from doctor to doctor in his anxiety for a "sure cure." Therefore, from the very beginning the physician must disclaim the possession of "sure cure" specifics and insist firmly, though sympathetically, upon an adequate program of treatment which he may hope will be successful. He must indicate that to attain satisfactory arrest of the disease a long time may be required. His winning of the patient's co-operation, faithfulness, and patience, is most essential. This is not an easy task. The patient's basic temperament (melancholic or buoyantly cheerful) modifies the attitude with which he launches on a program of rehabilitation. There enter also the psychologic reactions engendered by the disease and fixed by months or years of pain: resentment against his predicament, which has deprived him of occupation or security, resentment (sometimes tinged with shame) at the loss of self-esteem through physical deformity, and finally, often a profound resentment and distrust directed against the medical profession which, justifiably or not, may serve as a target for his discouragement over his present lot. Therefore, to assume the fullest responsibility for treating the arthritic patient, the physician must be an internist and at the same time something of a psychoanalyst and a psychotherapist. Seldom in the realm of medicine must he be so resourceful in the application of the artfulness of the physician.

The patient with arthritis may beseech the physician for compromises in the program of treatment, which, though decreasing the burden of sacrifice

for the moment may shatter the force of the whole therapeutic attack. Such compromises must be ruled out through sympathetic discussion. If that approach fails the physician must be firm in his insistence on what he deems necessary for meeting the situation half way may be worse than no treatment at all. Without treatment the patient may soon realize that he is getting nowhere and capitulate but if the physician accedes to the patient's demands for the easiest way there follows a period of months or maybe years during which the patient is holding to the hope that all will be well in the end while actually he is getting worse and developing irreparable damage. An arthritic cripple may thus be created. With his confidence in what medical science has to offer dissipated he may turn to unacceptable forms of therapy until through still more painful experience he learns his error.

Responsibility for what happens to these patients rests largely with the general practitioner. It must be granted that a small proportion of patients with an extreme degree of inherent susceptibility to the disease may develop literally malignant forms of arthritis which spread rapidly and devastatingly despite everything that may be done. Fortunately such cases are rare. It must also be granted that the socio-economic setting for the care of many of these patients is inadequate. However exclusive of these deterrents the chances for recovery from arthritis are predicated largely on the type of medical care that such patients receive at the outset.

The vast majority of patients with arthritis consult their family doctor early quite ready for any regimen that may be necessary to attain a cure. In the past we have not been very enthusiastic about the prospect of a cure and were inclined to approach the problem merely in the spirit that something should be done. It was an approach that was doomed to failure.

Successful management of arthritis demands a real interest in the problem, knowledge of every aspect of the disease and attention to plodding detail. To win the patient's confidence the physician must be wholeheartedly interested in the patient as well as in his disease. He must be thoroughly conversant with every phase of the situation so that he may speak with confidence derived from knowledge and experience. The challenge of the patient with arthritis to his physician is a challenge to the medical profession as a whole.

The physician cannot cope adequately with the problem unless he is qualified in the many fields of internal medicine that are involved either as an integral part or as concomitants of arthritic disease. Treatment of the many-sided constitutional background of this chronic condition (in which are involved hereditary predisposition, physical and nervous strain, fatigue, exposure, infection, disturbed function of the central and autonomic nervous system, of the peripheral circulatory apparatus, of the diges-

tive tract, and of the joints, plus numerous medical conditions incidental among arthritic patients) demands comprehensive knowledge and untiring effort. There is no "sure cure" for arthritis. A disease with so many wide spread implications cannot be effectively controlled by a vaccine, a diet, a series of sulphur injections, or any other single therapeutic agent. And yet a given means of treatment, inadequate when used exclusively, may be most effective when harnessed to other forms of therapy, all of which pulling together may reveal their strength.

SYSTEMIC TREATMENT

In general, the problem of treatment in the active stages of the disease is much like that presented by active tuberculosis. For, in addition to treating the local condition in the joints, the patient's general condition must be improved to combat the systemic effects of the disease.

Rest

Rest is undoubtedly the mainstay in the constitutional treatment of atrophic arthritis. It should include mental as well as physical rest—freedom from the cares of business and from every type of emotional stress. This prescription is more easily recommended than practiced, and is for this reason often neglected.

During the most active stages the patient must be kept strictly in bed. Ideal postural conditions in bed, which are highly important, may be best attained if sagging of the mattress is prevented and a small, flat pillow is used. If a hard, flat mattress is not available, boards placed under the mattress may prevent excessive sagging.

How long a period of rest is necessary is not always possible to predict. It depends on the severity of the arthritic process, on its activity, and on the resistance of the patient and his response to treatment. A minimum of several weeks of rest should be provided in the mildest cases, but, in more active atrophic arthritis, months may be necessary before the patient can safely be allowed out of bed. When the process is especially severe and active, and the response to treatment is slow, the period of rest may have to extend for a year or more. Whatever the length of time that may be required, no compromise is possible, for, just as cure of active tuberculosis may demand months or a year or two of complete rest, so may the cure of atrophic arthritis, in the last analysis, be determined by the length of time allowed for the complete physical rest that is so essential.

Only when the activity of the arthritic process has subsided may the patient be allowed to sit up, at first, for short periods during the day, and

then for increasingly longer periods. Even when the activity of the infection has largely subsided and it is in the more chronic stages sufficient rest must be provided so that fatigue is altogether avoided. When cure has been attained and the patient has been rehabilitated and has returned to work prolonged rest periods should still be provided. Such patients do well to interrupt the day's work sometime in the midafternoon in order to rest for at least an hour. We also instruct them to go to bed immediately after returning from work. By all means they must avoid working too hard and being too active physically; they must maintain optimum nutrition and avoid worry and nervous tension.

The misconception that confinement of a patient with arthritis to bed is tantamount to a sentence to ankylosis and invalidity is widely held by many physicians and patients and has militated greatly against institution of this most important measure of treatment. Patients say, "I have been told to keep going by all means if I want to prevent the joints from stiffening." So deeply entrenched is this belief that one often encounters active opposition when the suggestion is made that treatment must be started with a period of rest in bed. It is understood of course that merely putting a patient with arthritis to bed will not cure the disease. It should be equally obvious that if a patient were to lie in bed and neglect carrying out those specific measures necessary for maintenance of integrity of muscles and joint function, incapacity would probably result. But, when proper attention is accorded to the maintenance of joint function, nothing is more important in bolstering resistance for a cure in arthritis than is adequate provision for systemic rest.

Nutrition (The Diet)

There is hardly a form of dietetic therapy that has not had its vogue in the treatment of arthritis. What was lauded in one diet as the key to cure of arthritis was as vehemently disparaged in another. In general it seems that prohibition of some article of food in a diet scheme sufficed to lend therapeutic dignity to a dietetic formula and so from time to time meats, sweets, cereals, and so forth were prohibited. Fortunately, such prohibitions are gradually being abandoned.

Reduction of the carbohydrate allowance in the diet is in general based on sound principle: diets mordinately rich in carbohydrate are likely to be deficient in fruits and vegetables, vitamins and minerals. It also appears that there is better utilization of vitamins with diets restricted in carbohydrate. In general, however, the chief consideration should be to provide an adequate high caloric intake for those who are undernourished and a reduction in the caloric intake for those few who are obese. Otherwise the diet need not be different from that which would be prescribed if the patient

did not have arthritis. An abundance of fresh fruits and vegetables provides a high vitamin and mineral intake and is also valuable in preventing constipation. Various functional or organic disturbances of the digestive tract may dictate special modifications to suit the requirements in the individual case. In some extremely undernourished patients with appetites so poor that the food intake is too much curtailed, insulin is of value, since it may induce a gain of weight more promptly than would otherwise be possible. It is important to recognize, however, that for very actively ill patients a marked increase in the food intake may impose a load sufficient to strain the depleted capacity for metabolizing it. Generally, however, improvement is noted as the weight increases, and, in the author's experience, a sustained gain in weight has been a most favorable omen.

Vitamins

As we have already indicated, there is much reason to suspect that relative vitamin deficiency exists in many patients with atrophic arthritis. For this reason, we prescribe for such patients not only a high vitamin diet, but also various vitamin supplements. Vitamin B complex is routinely employed. When marked undernutrition, a poor appetite, marked disturbance of intestinal function, and perhaps dilatation of the colon exist, we feel that the parenteral administration of thiamin chloride (crystalline vitamin B) in doses of 5 to 10 mg., daily, or at longer intervals, for a time, is of distinct benefit. It appears that patients with such symptoms and marked vitamin B deficiency, *may be incapable of absorbing sufficient amounts of this vitamin administered by mouth*, parenteral administration of vitamin B₁ and of the vitamin B complex contained in liver extract insures an adequate supply of this material, and may bring about improvement in appetite and digestive function. After a preliminary period of such treatment, the administration of the vitamin B complex by mouth may prove equally effective.

Vitamins A and D are supplied routinely, generally in capsules of haliver oil and viosterol. Cod liver oil, in appropriate doses, is preferable, but unfortunately most patients have an aversion to cod liver oil and cannot be induced to take it.

Some clinicians have reported beneficial results obtained with large doses of vitamin D. We have given a small group of patients massive doses of vitamin D (40,000 to 100,000 units daily) but have found this form of treatment disappointing. Even if it were more effective, the practical difficulties encountered during the administration of massive doses of vitamin D over long periods of time would be a serious deterrent to its general adoption. We found, for example, that not infrequently gastric distress, nausea, loss of appetite, vomiting, and sometimes diarrhea were produced, necessitating either interruption of the treatment or its complete abandon-

ment The potentialities for harm from the administration of massive doses of vitamin D particularly the effects from hypercalcemia which may be induced must also be borne in mind

Since we are convinced that deficiency of vitamin C exists in many patients with active atrophic arthritis we frequently administer ascorbic (cevitamic) acid in doses of 50 mg several times a day, for several weeks Later a liberal allowance of fruits and fruit juices is depended upon to maintain adequacy of this vitamin in the body Vitamin C is especially to be supplied when there is a history of marked curtailment of fresh fruits and vegetables in the previous dietary or when symptoms suggestive of vitamin C deficiency exist that is bleeding gums a tendency to purpura, or to the development of spontaneous ecchymoses

Bowel Management

Constipation is a frequent complaint among patients with atrophic arthritis Exacerbations of the joint symptoms are occasionally accounted for by inadequate elimination It is doubtful whether constipation is in itself capable of causing so much harm Its correction should, however, not be neglected Drastic cathartics are unnecessary, proper diet, including sufficient roughage abdominal massage and exercises liberal intake of water and the addition of some intestinal lubricant usually suffice A mild saline cathartic is occasionally employed When there is spasticity of the colon tincture of belladonna may be of value Frequently it is combined with a mild sedative and is administered after meals The beneficial effect of the administration of vitamins particularly of vitamin B, is sometimes apparent by changes in the appearance and function of the colon Colonic irrigation may be of some value but only in certain selected cases in which colonic dilatation and stasis seem striking its employment routinely is not justified We have not observed any particular effect from implantation of bacillus acidophilus

Patients acutely ill with atrophic arthritis who are confined to bed may develop fecal impactions which may cause marked abdominal distress Although the possibility of this complication may be suggested by obstipation of several days duration it sometimes produces diarrhea which may confuse the diagnosis unless the possibility of fecal impaction is considered and rectal examination is made When impactions develop they must be broken up manually and treated in the usual manner

Blood Transfusions

During the early active or subacute phases of atrophic arthritis even when there is no anemia a series of small blood transfusions may be extremely beneficial They are particularly indicated and may be followed by dramatic

improvement when the arthritic process is very active or if the patient is markedly debilitated. In addition to increasing the hemoglobin and the red cell count, there is likely to be reduction of any existing fever, and sometimes striking diminution in joint swelling and improvement in appetite. In most cases there is also pronounced (although temporary) slowing of the sedimentation rate shortly after such transfusions. Recognition of this effect is important in evaluating determinations of the sedimentation rate obtained within short intervals after blood transfusions.

We administer from 250 to 500 cc. of blood each time, the amount depending on the weight of the patient and on the presence or absence of anemia. The first two or three transfusions may be given a week apart. Later, an interval of from four to five weeks may be allowed and additional transfusions then given if marked activity of the rheumatic process persists.

As we have stated, blood transfusions are most effective during the active, acute, or subacute stages of atrophic arthritis. They exert little influence on the process if given in the later, chronic stages of the disease. We have not employed blood transfusions primarily to correct secondary anemia for, in most cases, anemia responds well to systemic treatment and to the administration of iron and liver extract.

The precise manner in which blood transfusions serve to modify the activity of the rheumatoid process has not as yet been determined. In view of Davis' finding of hypoproteinemias in some cases, it may be that its beneficial effect is in some way connected with replenishment of the protein concentration of the serum.

Removal of Foci of Infection

If the eradication of focal infection is to be of value it should be accomplished during the early stages of the disease. The writer has seen cases in which the early removal of such foci was the determinant of recovery. The benefit from such operations decreases with the duration of the disease. Actually, the removal of focal infection late in the course of the disease will never arrest the process. Still, even in certain advanced cases of atrophic arthritis the removal of a definitely active focus of infection may relieve the systemic burden and improve the general condition of the patient to such an extent as to constitute an aid to recovery. In gauging what may reasonably be expected from the eradication of any etiologically related foci of infection, one must take a wide angled view of the entire systemic disease and of certain irreversible pathologic and physiologic abnormalities that may already exist.

One should not ignore the fact that certain risks may be entailed by operation. Removal of an infective focus may be followed by temporary exacerbation of the disease. The seriousness of such exacerbation can be

minimized by previous improvement of the patient's general condition and by postponing the operation until the most acute phase of the disease has passed. If the patient is in a seriously debilitated state, it is well to provide preliminary rest and to wait until some improvement in nutrition has been effected. Blood transfusion preceding the operation is sometimes indicated. It is impossible, however, to lay down hard and fast rules.

There is a most propitious time for operation if exacerbation of the systemic disease is to be reduced to a minimum. Experience aids greatly in determining the optimum time. One should aim, when possible, to remove infective foci when activity of infection in them is not acute, and when the patient's general resistance appears adequate to cope with the effects of the manipulations of the operative procedure. Finally, good operative technique is most in demand when the urgency for eradication of foci of infection is greatest.

Many considerations enter into the diagnosis and the evaluation of the relative importance of various foci of infection. This subject has been discussed elsewhere (Chapter VII). It should be stressed that the removal of foci of infection should never be relied upon exclusively for cure of the disease. The fullest benefit from this procedure is secured only when it is employed as a part of a well co-ordinated program of treatment.

Can foci of infection always be found in patients with arthritis? The belief that patients with arthritis harbor a focus of infection which, if found and eradicated, would promptly terminate the arthritic disease without further treatment presupposes that the arthritic process is a simple reaction between a given infective agent and an unfortunate set of articulations. This theory misses the wide implications of the systemic disease, inextricably involved in the complicated skein that is atrophic arthritis.

Although it is desirable to locate and eradicate all foci of infection related to the disease, this is, for various reasons, not always possible. The focus of infection may be beyond recognition or reach. Quiescent pelvic inflammatory disease, for example, if not extensive, may elude recognition by the most expert gynecologist only to reveal itself in a subsequent flare up. The author recalls a patient with focal arthritis, recalcitrant to treatment, even after the removal of all evident sources of focal infection, until the development of an acute perianal abscess led to the discovery of old anal cryptitis quiescent for a long time. The eradication of this source of infection led to an eventual cure of the arthritis with measures of treatment which previously had been unsuccessful. Meticulous examination should, of course, reveal such foci, but there is always the possibility of some hidden infection beyond reach of even the most careful observer. Then, too, related foci may be removed and residual areas left from which absorption of bacteria or their products may continue. The nasopharynx is possibly one such area,

which may persist even after perfect enucleation of the tonsils, and there may be other like situations. Obviously these areas are not amenable to surgical extirpation.

Again, eradication of the original sources of infection does not immediately suppress secondary foci that may have developed in joint structures or regional lymph nodes. If the importance of focal infection in the causation and maintenance of arthritic disease is actually as great as is supposed, we must extend the concept to include all possible secondary foci, particularly those in affected joints. Such foci, however, even if found, are beyond reach of surgical removal. Despite this apparently discouraging outlook the fact remains that treatment of such patients may still be amazingly successful, but the therapeutic attack under such circumstances must be comprehensive, recognizing the arthritic process in its entirety. General systemic treatment must be relied upon to attain the desired end, that is, improvement of the patient's general condition to a point where resistance is sufficiently high to cope with and even suppress the potentially dangerous residual infection. That is the only way through which, in the last analysis, any infectious disease is combated successfully.

SPECIFIC MEASURES

From time to time, new modes of therapy are brought forth—have their ardent exponents and, later, equally earnest and capable antagonists—and for varying periods each enjoys a vogue as a possible "specific" treatment for atrophic arthritis.

These measures have in the main been difficult to evaluate. There is, for one thing, a dearth of control studies by those initiating new forms of therapy in arthritis. The fact that atrophic arthritis may be a self-limited disease—it is certainly one subject to spontaneous remissions—further complicates the physician's efforts to appraise the value of new drugs. Disappointment with a new measure of therapy may engender distrust of all therapeutic efforts.

But newer measures of therapy in atrophic arthritis continue to enjoy increasing application once they have gained the momentum of active use. More often than not they are kept alive by intensive campaigns of advertising. Often glowing results of the use of these new measures are quoted out of their context. The "blurbs," so to speak, focus on the drug, not on the drug in relation to the entire medical regimen employed. For all these reasons the clinician finds himself at a disadvantage in choosing those resources most likely to be of value.

In order to consolidate the experience with certain forms of therapy—namely, colloidal sulphur, vaccines, sulfanilamide, fever-inducing machines,

and gold salts—a questionnaire was sent to the 178 members of the American Rheumatism Association, comprising physicians, all over the United States, particularly interested in rheumatic disease. Doubtless this cumulative experience constitutes an excellent cross section of clinical trial and serves as a touchstone to our own clinical experience.

One hundred and eleven replies to the questionnaire were received. Ninety-two physicians gave considerable detail regarding the value of these therapeutic measures. This group included physicians or clinics who had treated 200 or more cases with any given modality, as well as those who had treated only a few cases and thereby gained some clinical impressions as a guide. The other nineteen who replied did not furnish the data requested, either because they had not sufficient clinical experience, being interested only academically in the problem of arthritic disease, or because they were affiliated in the management of the same group of patients about whom their associates had reported.

For the present purpose detailed statistical analysis of this data is neither feasible nor desirable. In general the answers regarding the value of any given measure ranged preponderantly for or against. The data here collected (with the collaboration of Dr. V. W. Eisenstein) is a good indicator of the present status of each mode of therapy to be discussed. It may well serve as a guide to the perplexed physician who seeks unbiased knowledge on the value of certain 'specific' forms of treatment he should like to include in his armamentarium.

Colloidal Sulphur

A purely empiric notion led originally to the trial of sulphur in the therapy of arthritis. The experience with it has been extensive and the views of various observers who have reported on its value have been extremely contradictory. An excellent review of the literature is available in a report of the Council on Pharmacy and Chemistry of the American Medical Association (1938). This report constitutes a thorough, unbiased, and critical evaluation of the many isolated reports on the subject.

In 1937 I stated that sulphur injections had proved ineffective in my experience. The summary of the data from the questionnaires indicates that more physicians, who have given sulphur a trial, have abandoned its use than are continuing to use it (36 to 22). The more extensive the trial, the less favorable the impressions of this drug. Moderate users, who constitute the majority of reporters, found it "of no benefit" in arthritis, and (in a ratio of 7 to 1) report it "disappointing," "overrated," or "of no use." The dosage employed by both those who continued and discontinued its use was essentially the same. The occasional favorable reports appeared to be more enthusiastic than critical.

It is also significant, as the report of the Council on Pharmacy and Chemistry brought out, that all of the leading arthritis clinics in which sulphur therapy has been given a trial have abandoned it.

The detailed metabolic studies of Freyberg and his associates, reported only recently, are most illuminating. These authors emphasized the total lack of rationale in the therapeutic use of sulphur. Studying a group of patients with atrophic arthritis they could find no deviation from normal in the metabolism of sulphur. They showed that the concentration of sulphur in the blood of patients with arthritis is essentially the same as in normal control individuals. They demonstrated, further, that if a deficiency of sulphur existed in the tissues, this deficiency could not be remedied by administration of sulphur, since the drug is too rapidly and almost quantitatively excreted after parenteral injection or when given by mouth.

What, then, is the explanation for the favorable therapeutic results reported by some observers? Improvement following administration of sulphur was probably a coincidence, and not attributable to the drug. There is no questioning the psychologic effect of an injection—of any type of injection—in patients with arthritis. The beneficial effect ascribed to sulphur is probably to be ascribed either to a favorable psychogenic influence of the "injections" or to the well known tendency to spontaneous temporary remission in the severity of arthritic manifestations.

Vaccine Therapy

Various reports in medical literature can easily be assembled to support a brief either for or against the value of vaccine therapy in atrophic arthritis. The very number of vaccines for arthritis is sufficient proof that none is specific.

Despite an extensive experience with vaccines, Stansby and Nicholls (1933) were thoroughly disappointed with the results obtained by its use. Jordan (1937) *emphasized the need for controlled investigation*. He deprecated the routine use of vaccine with the "implication of certain cure." Only recently, the first controlled study on the value of "vaccines" and of "injections" in the treatment of arthritis was presented by Sidel and Abrams (1939) who found that while vaccine therapy is "beneficial" in 68 per cent of cases, sterile saline solution, similarly injected, is equally "beneficial in 72 per cent."

The promiscuous injection of vaccine in atrophic arthritis has probably caused more harm than good. There is little exact knowledge of how vaccines act in these cases. The experimental facts on which the various forms of vaccine therapy are based cannot be applied, in toto, clinically. Nothing could be more convincing of how sadly we are lacking in knowledge concerning the type and manner of vaccine therapy than the extremely

conflicting practices that have been advocated by various writers. As Holbrook stated: "There are investigators who report a large percentage of cures by giving millions of streptococci intravenously. Others, equally sincere, report similar results when the equivalent of less than one organism is used. There are ardent advocates of subcutaneous and of the intravenous method of inoculation. Stock vaccines are championed by some and decried by others. Agglutination, complement fixation and skin reactivity have all been defined as guides to diagnosis and therapy. Constitutional reactions are believed to be desirable or harmful depending upon the investigator."

From time to time we have studied various types of vaccine therapy. Undoubted clinical improvement attributable primarily to vaccine, is rarely observed and we have seen some unfavorable reactions. It is conceivable that a patient highly sensitive to streptococci can be harmed by subcutaneous injection of large doses of vaccine. Also, sensitization may possibly be created or increased by long continued administration of vaccine into the skin. Acutely ill patients are least likely to tolerate vaccine therapy; and, if their general resistance is low, a stubborn bombardment with vaccines may place them in serious jeopardy.

In the light of these facts let us analyze the clinical impressions of the value of vaccines in atrophic arthritis elicited by the questionnaire. 59 stated that they used vaccines, 24 that they did not, 8 that they used them only rarely, 14 that they had abandoned their use. Many definitely stated they employed vaccines much less frequently now than previously. About 60 per cent of users believe vaccines are beneficial to a degree. The consensus of opinion, however, indicated that fewer than 50 per cent of patients derived benefit from their use and these were for the most part early cases. The extent of usage varies considerably; in some clinics most atrophic cases are treated with vaccine while in others only those that fail to respond to other forms of therapy are so treated. Most popular are the streptococcus vaccines (used by 50 physicians), next the autogenous (41 physicians). Most workers emphasize the desirability of avoiding reactions. Of those who had not found vaccines of any benefit, most had given them extensive trial, had employed predominantly the streptococcic variety, and attributed whatever benefit was ascribed to vaccines by others largely to a psychogenic influence.

The author, through experience with various types of vaccines, was long ago led to conclude that the apparent benefit observed in the occasional case is largely attributable to a purely psychic effect. The results of Sidel and Abrams also confirm these conclusions.

Vaccine therapy, then, is anything but a panacea, its effect is not specific and its value limited. It may impress the patient that something tangible and specific is being done, but that is not true. It gives him a false sense of

security on which he may rely too much. If the periodic injection of vaccine be condoned on the score that it affords the physician an opportunity of seeing his patient often, and for long periods, and thus permits the application of other, important, therapeutic measures, it would be well that the physician duly recognize this fact. Too often though, the injection of vaccine becomes a fetish for the physician as well as the patient and other measures of therapy are neglected.

Fever Therapy

When physical means became available for the administration of fever therapy, it was hoped that fever inducing machines might be applicable to the treatment of atrophic arthritis. Extensive trial of this form of therapy was, therefore, carried out. Estimates of its effectiveness in atrophic arthritis have varied, depending, to an extent, on the enthusiasm of the observer. In general, the experience of others with this type of therapy has paralleled our own.

In 1933, we treated 13 patients with atrophic arthritis with hyperthermia induced by a high frequency current. Elevations of temperature from 103° to 104°F were induced, and then maintained for periods of from four to six hours, a series of four to six such treatments were given at intervals of a week. Seeking an opportunity for demonstrating the maximum benefit available from fever therapy, we selected patients who had had the disease for a relatively short period, and in whom the pathologic changes were confined largely to periarticular structures, with minimal degrees of bony change or deformity.

The initial improvement that generally occurred immediately after a 'treatment' was often remarkable. Stiffness and soreness generally disappeared, pain was abated, and, frequently, periarticular swelling disappeared completely. Naturally, greater freedom of motion and an increased sense of well being resulted. It was disappointing, however, that the gains were only temporary. Frequently within forty eight hours, practically always within a week, the condition of the joints resumed the status preceding the fever therapy session. All of the previous symptoms returned. Such initial improvement and subsequent recurrence of symptoms followed each session of fever therapy. We were equally disappointed to find no cumulative action from the series of treatments administered. Observing these patients for months afterward we were forced to conclude that there was nothing to recommend this form of therapy for atrophic arthritis.

Shortly afterward, Nicholls, Hansson, and Stainsby (1934) published their results on the treatment of this type of arthritis with hyperthermia. In 12 cases so treated their results coincided with ours in every respect. They, too, observed temporary relief of symptoms, but no lasting benefit.

They emphasized the fact that this form of therapy entails not only a trying ordeal for the patient but also some risk of complications. They concluded that their results did not justify continuing the employment of fever therapy in atrophic arthritis. Recently Krusen and Elkins (1939) indicated that 70 per cent of the patients with infectious (atrophic) arthritis treated by fever therapy exhibited little or no improvement. In the other 30 per cent who were improved the therapeutic program included, besides fever therapy, a well rounded medical regimen. Evidence in favor of fever therapy alone in this condition is obviously wanting.

The results reported through our questionnaire confirm the impressions just described. Thus, out of 92 reporting, 57 use, or have used, fever therapy as a modality in the treatment of atrophic arthritis. "Good results" were reported by very few, "poor results" were preponderant in the ratio of two to one. Reiterated in the replies were the dangers of fever therapy in all but the most robust patients. Two fatalities and two near fatalities were encountered. Many commented that the benefits from fever therapy were only temporary, that relapses usually occurred. The latter was the most frequent reason given for abandoning this type of therapy. The occasional good results reported were those in young subjects with early atrophic arthritis, or in sufferers from gonococcal arthritis.

These answers indicate that fever therapy at present holds little promise of benefit for the patient with atrophic arthritis. A combination of induced fever and some form of chemotherapy may possibly yield more worthwhile results in the future.

Sulfanilamide

In view of the possibility that a hemolytic streptococcal infection is related to atrophic arthritis, it seemed logical to try the effect of sulfanilamide in this condition. We administered this drug in adequate doses to a small group of patients with active atrophic arthritis. The results were entirely disappointing. Not only were beneficial effects not noted, but the reactions from the drug were for the most part disturbing, if not serious. Its use was therefore discontinued.

Reports of other observers confirm the impression we have gained. Swift, for example, found sulfanilamide valueless in the treatment of rheumatic fever, and Bauer and Coggeshall, treating ten patients with rheumatoid arthritis with large doses of sulfanilamide, observed no beneficial effect either on the clinical course of the disease or on the sedimentation rate. This contrasted with the beneficial effects observed in the treatment of gonococcal arthritis, in which there were both clinical improvement and reduction of the rate of sedimentation.

Sulfanilamide was tried by 44 of the 92 physicians who answered our

questionnaire The condemnation of this drug for the treatment of atrophic arthritis was practically unanimous It is clear that with present methods of administration sulfanilamide has no therapeutic value in this disease

Foreign Protein Therapy

We were at one time favorably disposed toward the use of nonspecific shock therapy with typhoid vaccine Further experience has led us to discard its use It has potentialities for harm, at times causing spread of the disease, and, at best, resulting in only temporary benefit A similar effect, *if desired, can be achieved with fever therapy applied by physical means*

Some clinicians are still employing foreign protein therapy in early or subacute forms of atrophic arthritis, believing that this form of treatment is especially applicable to mild, active cases with low grade fever Others reserve foreign protein therapy for the more indolent forms of atrophic arthritis Triple typhoid vaccine is most commonly used The stock vaccine, which in its original strength contains 2500 million organisms, is diluted so that 1 cc contains 50 million organisms The first injection usually consists of 25 million bacteria (0.5 cc), this dose is gradually increased by 25 to 50 million organisms, until a dosage of 500 to 600 million bacteria is reached Six to eight such injections are given intravenously at intervals of five to six days Some clinicians prefer to administer succeeding injections of typhoid vaccine twenty four to forty eight hours after the fever induced by the previous reaction has disappeared and the temperature has returned to normal The reaction is characterized by a chill, followed by a rise in temperature of varying degree, the latter depending not only upon the dose of vaccine administered, but also upon some inherent capacity of the patient for such a reaction

Bee Venom

Because of the belief that the sting of bees is a "cure for rheumatism" an injectable form of bee venom has been prepared and is recommended as being "of benefit" in atrophic arthritis Kroner and his associates reported that, of 100 cases treated with bee venom, 35 per cent showed "marked improvement" and an additional 38 per cent "moderate improvement" The author's experience with the same preparation was altogether disappointing

Chaulmoogra Oil

The beneficial effect ascribed to injections of chaulmoogra oil cannot be accepted, at present, as having been critically appraised Without comparative control studies one is inclined to wonder how much of the effect is due purely to the injection"

Gold Salts

Gold salts first recommended for use in arthritis by Forrestier, in France, have been employed rather widely in England and to a lesser extent in this country. Their mode of action in arthritis is unknown. They are certainly not a cure-all. Their use is sometimes attended by moderate, even serious, untoward reactions. However the importance of gold therapy in the therapeutic armamentarium for this difficult disease is enthusiastically attested by those with large experience in its use, even those who are most critical in evaluating their results. As Hench pointed out: "The curve of acceptance of most new treatments for arthritis that are destined to be discarded rises rather rapidly, reaches its peak in about three to five years, then falls as adverse reports begin to outnumber the optimistic ones. Finally, use of the treatment in any significant degree dies out after about eight to ten years." It therefore seems significant that the curve of acceptance of chrysotherapy is still rising after ten years of use.

The subject of chrysotherapy, which has received such merited comment in the European literature, has also been accorded favorable attention in recent American publications. It is the author's impression that chrysotherapy will be discussed more prominently and equally favorably in the future. In one way or another the physician will be seriously tempted to adopt this newer promising way of treating arthritis and therefore the present day knowledge on the subject will be described in some detail.

The most comprehensive study of every phase of chrysotherapy in arthritis including its toxic reactions is that of Hartfall, Garland, and Goldie (1937) who reviewed their results in 900 cases. No one who attempts to treat atrophic arthritis with gold salts should fail to review this report with the greatest care.

In any consideration of gold therapy, thought must be given to the danger of toxic reactions which constitute the most serious obstacle to the wide spread adoption of this promising measure. Unfortunately, the therapeutic use of gold is occasionally attended by certain serious complications, such as liver and renal damage, certain blood dyscrasias, and dermatitis, some of which end fatally. Yet, with increasing experience, it is becoming more and more evident that these toxic reactions are not entirely unavoidable, and that, to an extent at least, the frequency of serious reactions may be greatly reduced. Hartfall, Garland and Goldie reported 7 deaths directly attributable to gold out of 900 cases so treated, a mortality of 0.8 per cent. Most of these deaths occurred in the earlier days of chrysotherapy, however when larger doses of gold than are used at present were employed. It is noteworthy that, with reduction in dosage, the mortality rate was reduced from 3.1 per cent in their first 100 cases to the present figure of 0.8 per cent for the entire

series of 900 cases Copeman and Teguer (1937) encountered not a single fatality in their series of 51 cases treated with gold. It appears that by carefully modifying dosage and the method of administration, the mortality from toxic reactions can be reduced. The fact remains, however, that, no matter how useful, any drug potentially capable of inducing fatal reactions must be employed with utmost reserve.

Aside from the fatal reactions other toxic manifestations, appearing in a great variety of forms develop not infrequently. Thus, in the cases reported by Hartfall, Garland and Goldie, toxic phenomena, of greater or lesser severity, were exhibited by 42 per cent of the cases and, in 35 per cent of these, the reactions were more than trivial. Copeman and Teguer (1937) observed toxic reactions in 23 per cent of their cases, 17 per cent of which were mild, and only 6 per cent severe. The incidence of toxic reactions and their relative severity vary greatly in reports of different observers. But, no matter how carefully supervised this form of therapy may be, toxic reactions of some degree of severity are inevitable. Due recognition of their inevitability, in at least certain patients treated with gold is important. Such realization keeps the cautious clinician constantly on the alert for the appearance of mild reactions so that he may modify his course at the proper time and thereby avoid more serious consequences.

The toxic reactions from gold resemble those which occur with the use of salts of other heavy metals. The most common type of reaction attacks the skin and is manifest either by a scarlatiniform eruption, by an erythematous maculo papular rash, by pruritus, or, as occurs more rarely, by an exfoliative dermatitis. The latter is the most serious skin reaction and may result fatally. In general, however, these skin reactions are not serious. As a rule they appear during the course of treatment, although they may be delayed for weeks after it is completed. The rash usually persists for a week but may linger stubbornly for many months. In some of our cases herpes zoster appeared during the course of or shortly after, treatment. A variety of still other types of cutaneous disturbances has been described.

The mucous membrane of the alimentary tract may be involved by ulcerative stomatitis, which may cause soreness of the mouth. Although, such a reaction is usually transitory and seldom dangerous we have encountered cases in which the mouth ulcers did not heal for months and precluded the further use of gold. Nausea and abdominal colic occasionally develop shortly after an injection. We have never found this type of reaction to be severe, but serious, even fatal, cases of ulcerative colitis have been noted following the administration of gold sodium thiosulphate.

Like arsenic and phosphorus, gold may induce jaundice, caused apparently by a toxic hepatitis which, if severe, may produce permanent liver damage, or even a fatal issue.

Edema of the hands and feet or face, may occur. The latter appeared in one of our patients after the initial injection. Snyder (1939) reported the development of edema of the glottis which became so alarming that tracheotomy had to be performed. This patient had had a total of only 30 mg of gold sodium thiosulphate given in two doses intravenously. No doubt he had a high degree of inherent susceptibility to the toxic effect of this drug. Significantly this patient became completely free of arthritis symptoms after recovery from the toxic reaction and has remained so for the period of a year during which he has been observed.

Although one would not deliberately choose such a way of attaining cure of atrophic arthritis it is interesting that patients who experience severe toxic reactions frequently report striking amelioration or even apparent cure of the disease. We have occasionally noted focal reactions namely pain in the joints following injection sometimes discouraging to the patient at the moment but generally followed by rather striking improvement.

Blood dyscrasias of various types such as agranulocytosis, aplastic anemia and various grades of purpura may appear as toxic reactions to gold. Agranulocytosis or purpura developing with the administration of gold constitutes a serious complication and may prove fatal. Hartfall, Garland and Goldie (1937) reported the use of ascorbic acid in three of their patients who developed purpura and recovered. Whether the vitamin C administered facilitated recovery is not certain. These writers also reported the development of hypochromic and macrocytic anemia in patients under treatment with gold salts. We have observed the development of eosinophilia of 6 to 12 per cent in some cases. In none of these was the eosinophilia associated with any other toxic manifestation. Key (1939) (in whose series of 70 patients with rheumatoid arthritis treated with gold salts, 3 patients developed exfoliative dermatitis) reported that eosinophilia preceded the skin reaction in each case. Key therefore regards eosinophilia as a sign of an impending toxic reaction.

Albuminuria evidently as a manifestation of renal irritation, may develop. It occurs relatively infrequently, is generally a transitory phenomenon and may exist without the slightest impairment of renal function. True nephritis is surprisingly rare, we have never encountered it.

Unfortunately we have no way at present of determining which individuals are inherently hypersensitive to gold. Patients with an idiosyncrasy to the drug may develop a serious reaction after receiving the very first dose regardless of its size. In the majority of instances in which toxic manifestations develop however they are apparently caused by a cumulative toxic action of the drug. In such cases vigilance during the administration of

gold salts uncovers the early signs of mild toxicity, thus putting the physician on his guard against the development of the more serious manifestations

The presence of any of the common toxic reactions is no contraindication to further treatment at a later day. When multiple courses of gold therapy are used, toxic reactions tend to diminish in frequency during the second and subsequent courses. However, about 25 per cent of the cases develop toxic reactions in the second or later courses of gold therapy, even when *none occurred with the first course*.

Various means have been employed in attempts to prevent toxic manifestations. Hartfall, Garland and Goldie found that the use of calcium gluconate does not influence the incidence of toxic disturbances and have, therefore, abandoned it. We have routinely employed concentrated liver extract, and, more recently, ascorbic (cevitamic) acid in doses of 0.05 to 0.1 gm., injected simultaneously with each dose of gold salts but in a separate site, with the idea of preventing toxic blood reactions. Although eosinophilia and, occasionally, moderate grades of leucopenia have occurred in a proportion of our cases, serious blood reactions have not developed. In only two of our patients did hypogranulocytosis appear. It was asymptomatic and was detected by a routine blood count. Although it would seem that the use of liver extract or ascorbic acid may be worth while, the number of cases in which we have employed them is too small for any significant answer to the question of how much protection they afford.

Treatment of the toxic reactions from gold is largely symptomatic, nothing of specific value being available. The milder forms of skin reaction generally disappear spontaneously within a few days. Pruritus may be relieved by local application of calamine lotion, containing 2 per cent phenol. Purpura may be treated with large doses of ascorbic acid or liver extract. The latter is also useful when there is leucopenia or agranulocytosis, pentonucleotide has been used for the same purpose. In one of our patients who developed leucopenia with hypogranulocytosis, treatment with blood transfusions and liver extract promptly restored the normal blood picture.

A variety of preparations containing gold have been employed. In this country, gold sodium thiosulphate and sodium aurothiomalate (Merck) have been the preparations most readily available and most commonly used. In our earlier experience we employed gold sodium thiosulphate intravenously, in an initial dose of 0.05 gm. After several such doses, at weekly intervals, the amount was increased to 0.1 gm. and this dose was continued weekly until a total of 1.5 to 2 gm. had been administered. We then discontinued the drug for from two to three months, after which it was resumed, if manifestations of active arthritis or rapid sedimentation of erythrocytes were still present.

During the past two years we have employed gold sodium thiomalate (Merck) by intramuscular injection. The preparation is an aqueous solution which is preferable to oily suspensions, the absorption of which is likely to be variable. From the standpoint of ease of administration, intramuscular injection is of course preferable. In addition, we have gained the impression that our therapeutic results have been better with sodium auro thiomalate injected intramuscularly than they were when we employed gold sodium thiosulphate intravenously. Those with wider experience with chrysotherapy indicate however that the type of gold-containing preparation employed and the route of its administration are of no great importance in determining the results obtained, these being largely dependent upon the total amount of gold administered.

Hartfall Garland and Goldie recently (1938) reported the use of a new gold containing preparation (methyl glucamide of auro thio diglycollic acid) called Parmanal which they find to be superior to all preparations previously tried. They state that Parmanal is one of the least toxic of gold salts. In a preliminary trial the incidence of toxic reactions with this preparation was definitely lower and the curative results equalled or surpassed those obtained with other gold-containing preparations previously used even though the dosage has been approximately half of that previously employed. This drug may therefore represent an additional advance in chrysotherapy of atrophic arthritis. The preparation is not yet available in this country.

We generally start with a preliminary intramuscular injection of 0.01 gm of sodium auro thiomalate (Merck). The dose is increased from 0.01 to 0.02 gm and then to 0.025 gm, which is given at weekly intervals. A number of injections of 0.025 gm each are then given, and if this series is tolerated without reaction the dose may be increased to 0.05 gm weekly until a total dose of about 1.0 gm has been administered. In our cases in which mild transitory reactions appear in the course of treatment, the dose is not increased above 0.025 gm for any one injection. The therapeutic results in such cases are as satisfactory as those in which larger individual doses are used providing the total amount of gold given is adequate.

We inform our patients of the possibility of toxic reactions and indicate the most common ones that may occur. Before each injection the patient is questioned concerning the appearance of any reaction and the skin and mouth are examined. The appearance of excessive dryness of the skin or of pruritus is suggestive of the possibility of more serious skin reactions. The urine is examined at frequent intervals. We also study the blood count (at least the white and differential count) before each injection. A sharp drop in the number of leucocytes, a sharp increase in the proportion of non-granular leucocytes marked eosinophilia or combinations of these, are

warnings to interrupt treatment. These may seem to be unnecessary precautions, but it seems wiser to adhere to them until more safe, less toxic, gold containing preparations become available.

If only a mild reaction appears the dose of gold salts may be reduced or treatment temporarily suspended for a period of from one to four weeks, depending on the severity of the previous reaction. Re-examination of the patient at a subsequent time indicates whether gold therapy may be resumed. If upon resumption of gold therapy (generally with smaller doses than had been given prior to the appearance of the reaction) toxic manifestations reappear, this therapy is abandoned. Obviously, treatment is stopped immediately upon the appearance of any severe toxic reaction.

The sedimentation rate is determined several times during the course of treatment, always before and at the conclusion of a series. When the total dose of 1.0 gm. of sodium aurothiomalate (Merck) (0.5 gm. of gold) has been given, treatment with gold salts is discontinued and an interval of two to three months allowed to elapse. If, upon re-examination of the patient at the end of that time, indications of activity of the rheumatoid process are still present, as evidenced either by clinical signs, by a rapid sedimentation rate, or by an abnormal increase in the nonfilament, polymorphonuclear count, we advise that treatment with gold salts be resumed for a second course. At least two such courses of gold therapy are required in most cases. Hartfall, Garland and Goldie have administered three or four courses of gold (in one case five courses) before cure or marked improvement appeared. There is increasing agreement on the fact that individual doses of gold sodium thiomalate (Myochrysine) should not exceed 0.1 gm. and that the total for a course should not exceed 1.0 gm.

When the results of gold therapy (as reported by various observers with large experience) are analyzed, it becomes apparent that this is one of the most satisfactory adjuncts yet devised in the therapy of atrophic arthritis. Ninety-four per cent of the patients in Copeman and Tegner's series showed a favorable response to treatment, 58 per cent great improvement or quiescence of the disease. Ellman and Lawrence (1938) reported marked improvement in 88 to 94 per cent of their cases. Their report is particularly significant because the results of gold therapy were checked by a control series. It is to be emphasized that the results of treatment in this controlled study were estimated not only through clinical evidence, but also through determinations of the sedimentation rate, which generally showed similar improvement. In Hartfall, Garland and Goldie's series of 900 cases 67 per cent of the patients with atrophic arthritis who were able to complete the prescribed course of treatment were markedly improved or 'cured' and a further 19 per cent showed some improvement, a total of 86 per cent. These clinicians firmly believe that rheumatoid arthritis, if seen in its early

stages can be cured by gold and that there are few, if any, cases of the disease that cannot be improved to some extent

The author's results with gold therapy in 50 cases of atrophic arthritis have in general been equally satisfactory. The series is too small for detailed analysis of the degree of improvement, nor were these patients treated with gold alone. Nevertheless there is a clear impression that chrysotherapy contributed to inactivating the arthritis more often, more decisively, and more promptly than any other adjuvant measure of therapy previously employed.

Only 26 of 92 physicians who answered our questionnaire had used gold salts in the past or are using them at present. Most of these men are fearful of their use. They report reactions with approximately the same frequency as was reported from Europe: 10 per cent mildly toxic reactions, 25 per cent moderately severe, 0.8 per cent fatalities. Only one of this group reported a fatality—that from aplastic anemia. The hazard of toxic reactions from gold is implied, however, in the terse summary of one clinician: "used it once, never again." Most of those who employ it at present (and these have used it in 25 to 200 cases) are enthusiastic, believing gold salts to be the best single therapeutic measure to date in the treatment of atrophic arthritis as measured both by clinical improvement and by definite decrease in the sedimentation rate.

The value of gold salts in atrophic arthritis of the spine (Marie Strumpell disease) is still in doubt. It has been reported to be of value in the treatment of Still's disease. One of our patients with Still's disease, treated with gold sodium thiomalate, made a complete recovery and did not develop any toxic manifestations.

Gold therapy is particularly applicable to cases of chronic atrophic arthritis in which there is clinical evidence of activity of the process and a rapid sedimentation rate. It is of course useless in "burned-out" cases where the disease is inactive. Obviously, gold therapy does not affect directly the tendency to the development of joint deformities. It will certainly have no effect on old contractures, and will not restore the integrity of destroyed cartilage or bone. So far as the joints are concerned, the effect of gold therapy is most evident on the synovial and periarthritic tissues. Only to the extent to which gold permits the arthritic process to become inactive can it reduce the tendency to cartilage and bone destruction or prevent deformity.

It has been suggested that gold therapy be limited to cases of atrophic arthritis that have proved refractory to other forms of treatment. Our experience, as well as that of other observers, would indicate, however, that treatment with gold salts is particularly applicable to early cases in which the disease may be arrested before permanent damage has been done to cartilage or bone. Although more orthodox, conservative methods of treat-

ment without gold may yield satisfactory results and may inactivate the disease in a large proportion of cases, such improvement may be too long delayed, whereas, with the additional use of gold, arrest of the arthritic process may be accomplished in a much shorter period of time. This is, of course, an advantage, not only because it reduces the hazard of crippling deformity, but also because it permits rehabilitation of patients earlier than was possible heretofore.

Although we feel that gold salts represent a most useful adjuvant in the treatment of atrophic arthritis, there are good reasons for not recommending its more general adoption at this time. We feel that the potentialities for serious harm should always be considered. Patients presenting the slightest evidence of renal or hepatic insufficiency, or a tendency to purpura or leucopenia, should be rigorously excluded from chrysotherapy. Nor should the drug be employed in patients who have chronic skin affections, particularly eczema. Lupus erythematosus and psoriasis are exceptions, however, for gold has long been employed in the treatment of the former condition, and seems to be helpful also in the treatment of psoriasis. It is, moreover, too early to appraise the definitive place of gold therapy in atrophic arthritis. The practitioner who allows himself to be impressed with the glowing reports of the value of gold therapy to the point of substituting it for a "vaccine" or "sulphur preparation" as a "specific" for arthritis, undoubtedly has pitfalls before him. In using this modality one assumes the serious responsibility that goes with the use of a potentially dangerous drug. Moreover, whatever virtue chrysotherapy may have will surely be lost if it is not realized that this drug, like any other single measure of treatment in arthritis, is only a single link in a chain of therapeutic endeavors.

Drugs

Drugs occupy a relatively unimportant place in the treatment of any form of chronic arthritis. Analgesics for relief of pain may be necessary. If so, the salicylates are most satisfactory. They may be used freely, and are generally well tolerated. Although the effect of these drugs may not extend beyond symptomatic relief of pain, it is an end well worth seeking. Not only does it provide needed comfort and rest, but, with relief of pain, active exercise becomes tolerable. However, one must not depend upon analgesics alone for the relief of pain. Systemic rest, protection of inflamed joints by means of splints, and physical therapy—all of these, properly applied, may obviate the necessity for analgesic drugs.

Morphine should never be used for the relief of arthritic pain, addiction may be created too readily. "Rheumatism requiring morphine" should suggest the possibility of primary or metastatic malignancy (see page 519).

stages can be cured by gold and that there are few, if any, cases of the disease that cannot be improved to some extent'

The author's results with gold therapy in 50 cases of atrophic arthritis have in general been equally satisfactory. The series is too small for detailed analysis of the degree of improvement, nor were these patients treated with gold alone. Nevertheless there is a clear impression that chrysotherapy contributed to inactivating the arthritis more often, more decisively, and more promptly than any other adjuvant measure of therapy previously employed.

Only 26 of 92 physicians who answered our questionnaire had used gold salts in the past or are using them at present. Most of these men are fearful of their use. They report reactions with approximately the same frequency as was reported from Europe: 10 per cent mildly toxic reactions, 25 per cent moderately severe, 0.8 per cent fatalities. Only one of this group reported a fatality—that from aplastic anemia. The hazard of toxic reactions from gold is implied however in the terse summary of one clinician: 'used it once, never again.' Most of those who employ it at present (and these have used it in 25 to 200 cases) are enthusiastic, believing gold salts to be the best single therapeutic measure to date in the treatment of atrophic arthritis as measured both by clinical improvement and by definite decrease in the sedimentation rate.

The value of gold salts in atrophic arthritis of the spine (Marie Strumpell disease) is still in doubt. It has been reported to be of value in the treatment of Still's disease. One of our patients with Still's disease, treated with gold sodium thiomalate made a complete recovery and did not develop any toxic manifestations.

Gold therapy is particularly applicable to cases of chronic atrophic arthritis in which there is clinical evidence of activity of the process and a rapid sedimentation rate. It is of course useless in 'burned-out' cases, where the disease is inactive. Obviously, gold therapy does not affect directly the tendency to the development of joint deformities. It will certainly have no effect on old contractures, and will not restore the integrity of destroyed cartilage or bone. So far as the joints are concerned the effect of gold therapy is most evident on the synovial and periarthritic tissues. Only to the extent to which gold permits the arthritic process to become inactive can it reduce the tendency to cartilage and bone destruction, or prevent deformity.

It has been suggested that gold therapy be limited to cases of atrophic arthritis that have proved refractory to other forms of treatment. Our experience as well as that of other observers would indicate, however, that treatment with gold salts is particularly applicable to early cases in which the disease may be arrested before permanent damage has been done to cartilage or bone. Although more orthodox, conservative methods of treat

ment without gold may yield satisfactory results and may inactivate the disease in a large proportion of cases, such improvement may be too long delayed, whereas, with the additional use of gold, arrest of the arthritic process may be accomplished in a much shorter period of time. This is, of course, an advantage, not only because it reduces the hazard of crippling deformity, but also because it permits rehabilitation of patients earlier than was possible heretofore.

Although we feel that gold salts represent a most useful adjuvant in the treatment of atrophic arthritis, there are good reasons for not recommending its more general adoption at this time. We feel that the potentialities for serious harm should always be considered. Patients presenting the slightest evidence of renal or hepatic insufficiency, or a tendency to purpura or leucopenia, should be rigorously excluded from chrysotherapy. Nor should the drug be employed in patients who have chronic skin affections, particularly eczema. Lupus erythematosus and psoriasis are exceptions, however, for gold has long been employed in the treatment of the former condition, and seems to be helpful also in the treatment of psoriasis. It is, moreover, too early to appraise the definitive place of gold therapy in atrophic arthritis. The practitioner who allows himself to be impressed with the glowing reports of the value of gold therapy to the point of substituting it for a "vaccine" or "sulphur preparation" as a "specific" for arthritis, undoubtedly has pitfalls before him. In using this modality one assumes the serious responsibility that goes with the use of a potentially dangerous drug. Moreover, whatever virtue chrysotherapy may have will surely be lost if it is not realized that this drug, like any other single measure of treatment in arthritis, is only a single link in a chain of therapeutic endeavors.

Drugs

Drugs occupy a relatively unimportant place in the treatment of any form of chronic arthritis. Analgesics for relief of pain may be necessary. If so, the salicylates are most satisfactory. They may be used freely, and are generally well tolerated. Although the effect of these drugs may not extend beyond symptomatic relief of pain, it is an end well worth seeking. Not only does it provide needed comfort and rest, but, with relief of pain, active exercise becomes tolerable. However, one must not depend upon analgesics alone for the relief of pain. Systemic rest, protection of inflamed joints by means of splints, and physical therapy—all of these, properly applied, may obviate the necessity for analgesic drugs.

Morphine should never be used for the relief of arthritic pain, addiction may be created too readily. "Rheumatism requiring morphine" should suggest the possibility of primary or metastatic malignancy (see page 519).

Codaine too is rarely necessary but may be employed advantageously for short periods during the more acute crises as after manipulation of joints after operations and so on

Neocinchophen although a good analgesic is a dangerous drug because serious toxic effects especially liver damage occasionally develop from its use When there is a high degree of idiosyncrasy toward the drug liver atrophy may occur even with initial doses Aminopyrine too has recently been incriminated as a cause of agranulocytosis These drugs are therefore not recommended

Mild sedation is of value during the early phases of treatment Sedatives may also be required to aid sleep but if wakefulness is caused by aching in the joints aspirin taken before retiring may obviate the necessity for the use of sedatives Some patients are relaxed after the application of heat to the joints late in the evening just before retiring Others are stimulated by such physical therapy Obviously these must avoid heat late at night When large doses of sedative drugs are required to induce sleep one may be certain that emotional conflicts or other worries are the cause In this case it is useless to seek bigger and better sedatives the important thing is to find the cause of the insomnia

When there is anemia it is usually of the hypochromic type Any of the simple inorganic iron preparations (reduced iron ferrous sulphate or ferri ammonium citrate) given in adequately large dosage will correct it When the degree of anemia is pronounced liver extract may be given parenterally in addition to the iron Our employment of transfusions has not been primarily for the treatment of anemia

Dilute hydrochloric acid (at mealtimes) is indicated when the gastric acids are low or absent

Arsenic in the form of potassium arsenite sodium cacodylate or neoarsphenamine appears to be of value We sometimes employ it in the form of sodium cacodylate giving small doses by mouth

Iodides have no apparent virtue whether they be given by mouth or intravenously We have already stated that sulphur is useless and has no rational basis Orthoiodoxybenzoic acid (amyodoxyol or oxyoate) has nothing to recommend it It is not superior to aspirin

Endocrine Therapy

Unless there is evidence of an associated endocrine dyscrasia there is at present no rational basis for the use of endocrine preparations in atrophic arthritis A subnormal basal metabolic rate is not in itself an indication for the use of thyroid substance Desiccated thyroid may be of some value however in older patients who are obese and who present in addition to a low basal metabolic rate evidence of thyroid underactivity or of frank

myxedema Women who are harassed by menopausal symptoms, should be relieved of the vasomotor instability and increased nervous tension by the administration of estrogenic substance in adequate dosage

The observations of Hench, and Sidel and Abrams on the inactivating effect of jaundice in arthritis hold a challenge to our resourcefulness in devising some biologic or chemotherapeutic agent to aid in controlling some phases of the arthritic process Stimulated by these observations, we have studied the effect of administering sodium dehydrocholate (decholin sodium), a salt of one of the bile acids, but found it ineffective Reasoning that the effective agent may be a product of hepatic degeneration, we have also tried the intravenous injection of an autolysate of liver but again with out encouraging results

The relief of arthritic pain afforded by the icteric state is probably effected not by any one single chemical factor, but possibly by some combination of factors inherent in the state of jaundice Attempts made so far to reproduce the phenomenon (Hench, Thompson and Wyatt) have brought only equivocal results which are not yet applicable in practical therapeutics Nevertheless, we cannot relinquish the idea that eventually some component of the icteric state may be discovered which will duplicate nature's own therapeutic success

"New" Remedies

New drugs and forms of treatment for arthritis are constantly being exploited Some of these new remedies are the products of research which has not been sufficiently substantiated to warrant acceptance In evaluating new therapeutic measures it is well for the physician to ascertain that the source of the remedy is reliable, that the rationale of the treatment is sound, and that the suggested reason for its effectiveness is consistent with the known basic principles underlying the rheumatic process

In general, it would be better if the physician placed his chief dependence on those tried measures on which he may safely rely, leaving to research clinics the study of newer therapeutic measures If the newer therapeutic experiments in arthritis are employed it should not be at the sacrifice of the gains to be obtained from the more orthodox, more reliable methods, because flitting about from one new "panacea" to another is almost certain to cost the patient unnecessary loss of joint function

CLIMATOTHERAPY

A warm, dry climate with minimal fluctuation in barometric pressure is ideal for the patient with arthritis Along with these favorable atmospheric conditions there goes the availability of heliotherapy which, if properly

applied may be of additional value. Time spent in a suitable resort is obviously worth while for those who can afford this luxury, but one must not be deluded into the belief that a change of climate alone is the key to cure. A patient who recovers from arthritis at a resort or spa might have recovered equally satisfactorily at home. Such advantages of climate are furthermore available to relatively few persons. To most others it adds an economic burden which more than detracts from the benefits that might otherwise be attainable. Climatotherapy is therefore not to be chosen by the physician as the way out of a difficult problem presented by the chronic arthritic invalid.

OPERATIVE TREATMENT

In carefully selected cases presenting marked peripheral vasomotor disturbances and free movable joints sympathectomy may by permanently increasing circulation serve as a useful procedure. This operation however is applicable to only a very small proportion of cases. It might be justifiable for patients whose arthritis is limited to the hands or feet. Interruption of the sympathetic innervation may relieve them of the discomfort caused by cold clammy extremities. By producing vasodilatation it is equivalent to providing a sort of perpetual baker. But such operations obviously exert no other effect on the fundamental factors entering into the process.

Bone drilling of the epiphyses adjacent to affected joints has been recommended by Forbes Mackenzie and others for both atrophic and hypertrophic arthritis. Critical analysis of the results creates a doubt as to whether the benefits obtained are attributable to the long periods of rest incident to this operation or to the surgery itself. In hypertrophic arthritis in which improvement may be more noticeable the possibility exists that this procedure actually increases the circulation to the affected joints.

Operations on the joints proper which aim to correct deformity and increase the range of function are important for the rehabilitation of many patients with arthritis. This aspect of treatment is discussed in detail in the sections dealing with the management of deformities (page 167).

PSYCHOTHERAPY

Regardless of what form of therapy is employed the patient needs understanding of the problems and treatment of his disease and encouragement with regard to the results that may be expected. The influence of a multitude of psychic factors on the course of atrophic arthritis is recognized and met too seldom. The clinician must recognize the emotional conflicts that often arise in this disease as a significant part of the patient's condition and

he must attempt to communicate to the patient some sense of emotional security. The nervous and psychic state of the patient and its competent handling may determine to a large extent the outcome in any serious case of atrophic arthritis.

It is important that the physician maintain an attitude of balance and sympathy toward the many problems of his patient. With experience, he learns to anticipate the many questions that perplex the patient with arthritis—the feeling of insecurity about the outcome, the doubts that arise as to the efficacy and the wisdom of what is being done, anxiety about the future, and so on. The physician must be prepared not only to minister to the patient's physical ills, but also to act as his patient's counselor and advisor. He has reason for optimism, an attitude entirely justified by the results that may be attained. But, in prognosticating the probable outcome in certain cases, he must be realistic, he must avoid exaggerated predictions of what may be accomplished.

Irreparable physical damage must be accepted as the liability that it is. But despite such liabilities the patient may retain worth while, perhaps invaluable, inner resources which he should be induced to use to the greatest advantage, as Professor DaCosta and Clarence Day used their gifts, one as a great teacher and the other as a tolerant social satirist.

PREVENTION

Preventive measures, which, it would seem, should be given first consideration with reference to the control of any disease, have too often lagged far behind the application of purely remedial measures. This is particularly so in chronic rheumatism. So long as we do not know all of the specific factors that enter into the causation of chronic rheumatism, it is, admittedly, impossible to lay down specific, hard and fast rules for its prevention. Nevertheless, as we have indicated in the introductory section, there is strongly suggestive evidence that certain physiologic and pathologic mechanisms are related to the rheumatoid syndrome. A number of these are subject to our control.

We are obviously handicapped in altering in any way an inborn constitutional susceptibility to atrophic arthritis. We can, however, alter, or at least modify to some extent, those environmental factors which are known to enhance this basic rheumatic tendency. Thus, in the prevention of chronic rheumatism, it is very important to maintain optimum nutrition, adequacy of vitamin supplies, a well poised and integrated nervous system free of excessive strain, and to avoid overwork. It need hardly be said, even parenthetically, that many of these measures are, in turn, conditioned by social and economic circumstances over which society, and not the physician, has

control In view of the apparent relationship between focal sepsis and atrophic arthritis it would also seem logical that preventive medical measures should include provision for the early eradication of focal sepsis preferably before organic joint disease has set in In order that attention may be directed to these factors at the proper time the physician must examine the patient carefully and pay scrupulous attention to the correction of every abnormality he finds in the course of the routine periodic physical checkup Preventive measures are to be especially applied among those individuals who present some hint of the rheumatic constitution either because of an obvious familial disposition to the disease as revealed by the history or because of the presence of those stigmata of the arthritic constitution we have already discussed

PROGNOSIS

It is not always possible to forecast the eventual outcome of the disease In a small proportion of cases the patient's inherent susceptibility to it or other unfavorable circumstances may lead to crippling and life-long invalidism On the other hand there are mild cases in which despite the inadequacy or lack of treatment sufficient natural resistance is mustered to combat the disease Between these two extremes are many other patients seriously affected with widespread involvement of joints who may under ideal circumstances make either a complete recovery or achieve arrest or quiescence of the process and remain reasonably free of disabling manifestations

The outcome is determined to a great extent by the adequacy of treatment during the early stages of the disease That depends in turn on how interested and how well informed the physician is who sees the patient at the onset of the arthritis It depends also on whether the physician and patient will submit to the requirements of logical therapy or whether they will angle constantly for some quick sure-cure It is not easy for the patient with arthritis to accept the rigorous discipline imposed by this disease He must learn to do so however and the physician can help by teaching him how The patient must have not only the desire but the will to get well yet he must guard against tension over the battle lest he become worn out

The facts pointing most clearly to what is being accomplished are the actual results Careful evaluation of clinical records by competent authorities with large experience in the treatment of arthritis reveals the encouraging fact that 75 to 90 per cent of the patients with chronic arthritis may be helped either to complete recovery or to definite improvement Approximately 25 per cent have been found to recover completely This is an impressive figure especially in the light of the fact that most of these patients

were in the advanced stages of the disease. Often such patients had run the gamut of therapeutic measures before consulting a physician interested in arthritis. The results of thorough treatment of patients with this disease, including those with less severe forms, would undoubtedly reveal a much higher proportion of very gratifying results. The impression that arthritis is an incurable disease must be revised. On the contrary, there is much reason for hope for the patient with chronic arthritis. The road to recovery may be long and devious, it is often slippery, but it is one which today may be taken with confidence.

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PART II

PREVENTION AND CORRECTION OF
DEFORMITIES IN CHRONIC ARTHRITIS

AN ILLUSTRATED GUIDE TO THE RECOGNITION OF THE CHARACTER AND CAUSES OF ARTHRITIC DEFORMITIES AND THE MEANS FOR THEIR PREVENTION AND CORRECTION

PHYSICAL THERAPY IN ARTHRITIS

CHAPTER XIV

THE PREVENTION AND CORRECTION OF DEFORMITIES IN CHRONIC ARTHRITIS

Throughout the ages nature has cured many cases of arthritis, the infection burning itself out in time, but leaving behind painless deformed derelicts. If the pain of the disease has been alleviated the deformities with their consequent discouragement, have persisted. We may question not without justification, the gain in the patients' recovery from the systemic infection if they are to be preserved for a life of helplessness.

Suppression of the systemic process is not enough. No matter how well chosen and how carefully applied a program of treatment for arthritis may be, it will fail in its purpose—that is the restoration of a normal individual if it does not achieve two general objectives: quiescence of the inflammatory process and, while this is being done, prevention of deformity.

Deformities in arthritis are not inevitable, they do not just occur, they develop because someone has not known how, or has not exerted the effort to prevent them. And once they have occurred their correction is difficult, if not impossible. Hence the emphasis on prevention.

One must be particularly alert to prevent chronic deformities in atrophic arthritis where the tendency to deformity is initiated by the earliest pathologic changes in the periarthritic tissues, changes which, unrelieved, become increasingly more pronounced and more damaging. We have effective means of combating the threat of arthritic deformities, and, if these therapeutic weapons demand exacting laborious application, it is no reason for not making the most of them. The labor will be more than rewarding.

Steering an arthritic patient through the many vicissitudes of the systemic infection, for months or years, deserves better results than those generally attained in the past: the cured patients with dreadful deformities of various grades, with bent and partially fixed knees, with distorted and painful feet, and with ankylosed wrists, fingers, or elbows. In the past the failure to prevent these deformities has helped to swell the appalling number of arthritic cripples. The histories of such patients often reveal that these deformities developed and progressed so insidiously that the patient and the physician are frequently unable to recall when they actually began.

To understand the rationale of the program of therapy aiming at the

prevention of deformities one must have clearly in mind the train of disturbances constituting the pathologic physiology in the joints in atrophic arthritis the factors which bring them about and which perpetuate them

Three factors in atrophic arthritis operate to produce joint damage the painful muscle spasm atrophy of muscles and contracture of periarticular tissues and possible fibrous union and ankylosis through proliferation of granulation tissue and destruction of cartilage There has been a tendency in the past to look upon these occurrences as inevitable That is decidedly not the case Actual experience shows that the harmful influence of these factors can be modified or entirely prevented in almost every case save those exceptional instances in which the destructive process within the joint is a fulminating one from the start

The driving force leading to the damaging deformities is pain for pain about the joints and soreness of the muscles are the dominant factors controlling the function of the articulations during the active phases of atrophic arthritis To splint the inflamed and painful joints to relieve them of the added pain of motion there develops spasm of the surrounding muscles particularly always the flexor groups spasm which is involuntary and incessantly maintained Exudation and swelling within the joint as well as in the periarticular structures aggravate this tendency to inactivity

Unmolested the patient is grateful for whatever relief from pain is afforded him in this way but with time the spastic flexor muscles become atrophied and contracted motion becomes more painful hence more limited and soon all the sequelae of deformity are evident Thus are produced the flexion deformities and even subluxations at the joints With the joints in abnormal positions contracture of the capsular structures and atrophy of the muscles prevent resumption of normal alignment at the joints the deformities being thus maintained and perpetuated When weight bearing joints of the lower extremities are involved the strain of weight bearing if not eliminated adds its share to exaggerating the deformity by producing additional injury through traumatic insults on joints in functionally abnormal alignment Meanwhile, the inflammatory synovial pannus within the joint and the destructive changes in the articular cartilage are proceeding toward permanent ankylosis

To prevent these deformities the physician must anticipate nature's attempts to secure physiologic rest for the diseased articulations Fixation by splinting may be entirely adequate for those joints of the upper extremities which do not bear body weight but for those of the lower extremities which in addition to the function of locomotion are burdened also with carrying the weight of the body relief from that burden by rest in bed becomes an indispensable necessity

Prevention of serious damage to inflamed joints is possible at the earliest stages of the disease, a little later it may be too late. The idea of the patient, bolstered, unfortunately, by the advice so often given him, "to keep walking no matter how much it hurts," is the shortest route to the wheel chair. Recovery from arthritis sometimes occurs despite full activity, but not because of it. On the other hand, the ankyloses that develop in patients, while in bed, cannot be attributed to the bed rest, they are generally the result of improper medical supervision and treatment, or of accepting confinement too late, that is, only when forced into inactivity by too drastic destruction of the articulations, or by deformity.

It is not generally realized what relief from pain can be afforded the sick arthritic patient by merely splinting all of the inflamed joints and relieving them of the strain of unguarded movements and of constantly maintained muscle spasm. When such splinting is combined with daily exercises, thus not permitting complete immobility for too long stretches at one time, prevention of contracture of muscles and capsule, and of ankylosis, is practically certain. It is the author's belief that if every inflamed joint in these patients were moved, either passively or actively, at least once a day, through the fullest arc of its normal range of motion, ankylosis of joints would be the rarest sequel of arthritis.

On the other hand, permitting a patient unlimited use of his inflamed, sick joints, and particularly weight bearing joints, probably accounts for a large share of the inflammatory reaction and damage that we now see in arthritis. This is easily proved by observing how quickly some resolution of exudation, swelling and pain occurs when an actively ill patient is merely put at rest and the joints immobilized. The more acute the inflammatory process and the more recent the arthritis the more striking is the evidence of improvement on relief from physiologic trauma. No doubt, the severity of an arthritic process is actually much exaggerated before this extrinsic traumatic factor is eliminated. I prefer, therefore, to estimate the severity of a given arthritic process after a short preliminary period of observation during which time the patient is confined to bed and the joints are appropriately protected from extrinsic insults of even physiologic trauma. Such an estimate is always more accurate, and frequently more encouraging, than that of the initial examination at the office. The situation is comparable to that of the patient with diabetes, who, when he first presents himself for treatment, may exhibit a dangerous state of marked hyperglycemia, glycosuria, and acidosis—largely the result of excessive carbohydrate intake and cumulative extrinsic strain on his glycogen metabolism—but who after a few days' treatment, with appropriate restriction of carbohydrate and administration of insulin, turns out to have a relatively mild diabetes easy to control.

It is generally impossible to predict for how long a period of time rest of the joints must be carried out. Several months may be the minimum though when the arthritis is severe a year or two may be required. It is a safe general rule that the earlier rest is resorted to the less will be the time required. This phase of the treatment injects a problem which in practice is not always easy to surmount. The patient in the early stages of his rheumatic affection is not likely to accept confinement to bed readily, thinking his joint involvement too trivial for such a drastic measure. He may argue that such confinement spells economic hardship which to many it actually does but most often the patient indicates that he wants to fight his disease with valor. Regardless of distress from pain he wants to keep going as he says to keep the joints from stiffening up. In this way he hopes he will not repeat the awful spectacle of the patient he knows who kept going with arthritis for years but once submitting to confinement to bed has not left his wheel chair or bed to walk again. He will argue that he will do anything if you will but waive that injunction that he remain in bed. I will follow strictly any diet. I will come to your office for treatments as often as you wish he will implore but do not insist on my going to bed. And as we have already hinted his arguments may be genuine enough. The economic hardship imposed on the breadwinner or the housewife by confinement to bed may be serious deserving the physician's utmost consideration and sympathy. Many of these patients have no one to care for them or to take their place in the home.

Facilities for hospitalization of the vast number of patients with arthritis is now woefully inadequate and the cost of hospital facilities for long periods is beyond the reach of many of them. For these patients adequate facilities for home care must be improvised and facilities for institutional care enlarged.

PROCEDURE FOR THE PREVENTION AND CORRECTION OF DEFORMITY

What procedure specifically will prevent excessive joint damage and deformity when instituted at the very start of the disease? This is the phase of arthritis with which the family physician is chiefly concerned. At such time he is the one most likely to be called upon for treatment.

At the onset there is pain, soreness in the joints and muscles, probably some degree of periarticular swelling and muscle spasm. The patient himself is anxious almost exclusively about the relief of pain. He rarely suspects the graver implications of his disease. The physician though must not be content with the mere relief of pain. He must seek to place the joints as well as the patient in a position which will afford maximum protection.

from further damage to joint structures, that will aid resolution of the inflammatory process in the joints, prevent further muscle spasm and resolve that which is already present, maintain the best integrity of the musculature, preventing its atrophy and contracture, and, by doing all of these things, prevent flexion deformities. To accomplish all of this, rest for the inflamed joints is the primary essential, but it must be carried out in such a way that it maintains the physiologic integrity of the articulations, for a thoroughly healed joint that does not work is small consolation to the patient.

Rest for inflamed joints may be provided either by relieving them of functional strain or, when there is muscle spasm, by fixation in splints or plaster casts which may be cut to provide properly fitting, removable plaster shells. We have also employed cellulose compound casts for such fixation.* They have proved extremely satisfactory, they are much lighter in weight than plaster casts, are washable and durable, and, because of greater resiliency of the material, permit easy removal when necessary.

The joints are fixed as far as possible in the position of extension. During the most active phase of the inflammatory process, these supports may be left on for two or three days. They should then be removed and passive or active motion instituted for a few minutes each day, with complete rest between times. Motion is carried out slowly, deliberately, within the limits of pain. Active motion is the most important although assistive support may extend the degree of mobility. Active exercises are increased in range and frequency as conditions permit, depending largely on the absence of pain and protective muscle spasm. While motion in the joints should be attempted as early as possible, it should not be overdone when the process is most acute, as excessive motion at this time may increase the activity of inflammation in the joints. Gradually, the splints are dispensed with for longer periods, during which time baking, light massage of the muscles, and active exercises are practiced. Later, group muscle exercises are extended by appropriate occupational therapy. But even when acute activity of the process has largely subsided, the use of splints at night may be indispensable to prevent spasm, flexion and contracture of muscles during sleep.

What can be done when the joints have already begun to flex, that is, when the first chance of preventing deformity has been missed, depends on the degree of deformity present and the cause of it. If the extent of the deformity is only slight, flexion having only recently begun as a result of muscle spasm and early muscular contracture, correction may still be a very simple matter. Merely splinting such joints in the position in which they are found, and maintaining immobilization for a day or two, may relax

* The bandage used in making these casts is distributed by Baucor & Black, New York, and Lewis Manufacturing Co., Walpole, Mass., under the trade name of 'Caster'.

muscle spasm sufficiently to permit some straightening of the joint when the splint is removed and physiotherapy applied. If this happens and a few degrees of extension have been gained a new plaster splint may be applied in the corrected position and with continued rest heat massage and exercises further correction may be possible. This procedure may be repeated indefinitely until full relaxation of muscle spasm is effected and with it full restoration of normal alignment at the joints. This type of corrective splinting is applicable to almost every joint.

In appropriate cases the correction attained may be maintained with skin traction for a time in order to induce the fullest degree of stretching of previously contracted flexor muscles.

When the deformity is more pronounced in degree is of longer duration is already maintained by shortening of the responsible muscles—generally the flexors—and is complicated further by contracture of the periarticular capsule rest alone will induce little change in the degree of deformity. Some extrinsic force must now be added to effect straightening of such joints. This force may be supplied by (1) gradual manual manipulation over an extended period of time combined with physiotherapy (2) skin traction (3) wedging of casts (4) forcible manipulation with the patient under anesthesia (5) application of skeletal traction and (6) open surgical procedures.

1 *Gradual manual manipulation* may in time be thoroughly effective in attaining complete correction of minor grades of deformity if the manipulation is performed by one experienced in the method that is one who knows how far to proceed each time to make the maximum gain without throwing obstacles in his own way by overtaking the capacity of the joint by too much trauma and the morale of the patient by too much pain. When correctly carried out such manipulation yields consistent though small gains each time. Through the cumulative effects of the manipulative treatment and the concomitant splinting traction physiotherapy massage and exercises complete correction of the deformity is brought about as well as improvement in the functional integrity of the musculature. Although on the whole this is a slow plodding manner of correcting deformities it is an eminently satisfactory one from the standpoint of the functional result that may be obtained. And in the last analysis the functional capacity of the joints after correction is what counts the most.

2 *Skin traction* is applicable chiefly to knee flexion deformities of mild severity caused exclusively by muscle spasm or contracture. It has the advantage of acting constantly but at the same time permitting readily the application of physical therapy. Traction is instituted in the line of deformity and because this changes with improvement it requires frequent realignment.

Banjo splints are an excellent means of exerting traction for correcting deformity in the fingers and hands, and may be employed to better advantage following initial manipulation under anesthesia

3 *Wedging of casts* is one of the commonly employed methods for the correction of flexion deformities. A plaster cast is applied to the limb, the cast being then divided on the flexor surface and wedges inserted so as to force the limb into extension progressively. There are some disadvantages, such as the possibility of posterior subluxation of the tibia when knee flexion deformity is pronounced, and also the more remote danger of fracturing of demineralized bone

Turn buckles may be employed with plaster casts instead of wedges, particularly for the elbows and knees

4 *Manipulation of joints*, with the patient under anesthesia, is a method well adapted to the correction of deformity caused by moderate degrees of muscular and capsular contracture, and is effective in breaking up of intra articular and periarticular adhesions which would not yield to the more gentle, gradual manipulation already described. This procedure has been accorded wider employment, with greater success, since its advantages and limitations have become more specifically known. Obviously, such forcible manipulation, with the tearing of adhesions, may result in some flare up of activity of the arthritic inflammatory process. It must not, therefore, be attempted at the height of activity of the disease if one is to avoid increased destruction of intra articular tissues and ankylosis. However, if more conservative measures tried over a period of weeks or months are ineffective in reducing the degree of deformity, and the arthritis is essentially quiescent, it is not necessary to wait until all activity of the arthritic process has ceased

On the whole, the advantages of manipulation outweigh the risk of the procedure even in the face of some residual activity of the arthritic process. The possibility of temporary activation of the inflammatory process is to be feared less than the danger of firm fibrous ankylosis because of delay in undertaking manipulation. It must be remembered, too, that atrophy progresses as the joints remain immobile. In any event, however, one should anticipate some degree of activation of the process and be prepared to employ such measures as would aid in combating this effect

We frequently give blood transfusions after manipulation, particularly when there is any significant rise in temperature or acceleration of sedimentation rate, and have found them of distinct value. The joints are treated by splinting in the corrected position, and physiotherapy as well as exercises are instituted as early as possible

Manipulation is not entirely free of potential hazards, such as excessive damage to joint structures, rupture of blood vessels, and even fracture of

muscle spasm sufficiently to permit some straightening of the joint when the splint is removed and physiotherapy applied. If this happens, and a few degrees of extension have been gained, a new plaster splint may be applied in the corrected position and, with continued rest, heat, massage, and exercises further correction may be possible. This procedure may be repeated indefinitely until full relaxation of muscle spasm is effected, and with it, full restoration of normal alignment at the joints. This type of corrective splinting is applicable to almost every joint.

In appropriate cases the correction attained may be maintained with skin traction for a time, in order to induce the fullest degree of stretching of previously contracted flexor muscles.

When the deformity is more pronounced in degree, is of longer duration, is already maintained by shortening of the responsible muscles—generally the flexors—and is complicated, further, by contracture of the periarticular capsule, rest alone will induce little change in the degree of deformity. Some extrinsic force must now be added to effect straightening of such joints. This force may be supplied by (1) gradual manual manipulation over an extended period of time, combined with physiotherapy, (2) skin traction, (3) wedging of casts, (4) forcible manipulation with the patient under anesthesia, (5) application of skeletal traction, and (6) open surgical procedures.

1 *Gradual manual manipulation* may, in time, be thoroughly effective in attaining complete correction of minor grades of deformity, if the manipulation is performed by one experienced in the method, that is, one who knows how far to proceed each time to make the maximum gain without throwing obstacles in his own way by overtaking the capacity of the joint by too much trauma and the morale of the patient by too much pain. When correctly carried out, such manipulation yields consistent, though small gains each time. Through the cumulative effects of the manipulative treatment and the concomitant splinting, traction, physiotherapy, massage, and exercises complete correction of the deformity is brought about, as well is improvement in the functional integrity of the musculature. Although on the whole, this is a slow, plodding manner of correcting deformities, it is an eminently satisfactory one from the standpoint of the functional result that may be obtained. And, in the last analysis, the functional capacity of the joints after correction is what counts the most.

2 *Skin traction* is applicable chiefly to knee flexion deformities of mild severity, caused exclusively by muscle spasm or contracture. It has the advantage of acting constantly but at the same time permitting readily the application of physical therapy. Traction is instituted in the line of deformity, and because this changes with improvement, it requires frequent realignment.

Banjo splints are an excellent means of exerting traction for correcting deformity in the fingers and hands, and may be employed to better advantage following mitral manipulation under anesthesia

3 *Wedging of casts* is one of the commonly employed methods for the correction of flexion deformities. A plaster cast is applied to the limb, the cast being then divided on the flexor surface and wedges inserted so as to force the limb into extension progressively. There are some disadvantages, such as the possibility of posterior subluxation of the tibia when knee flexion deformity is pronounced, and also the more remote danger of fracturing of demineralized bone

Turn buckles may be employed with plaster casts instead of wedges, particularly for the elbows and knees

4 *Manipulation of joints* with the patient under anesthesia, is a method well adapted to the correction of deformity caused by moderate degrees of muscular and capsular contracture and is effective in breaking up of intra articular and periarticular adhesions which would not yield to the more gentle, gradual manipulation already described. This procedure has been accorded wider employment, with greater success, since its advantages and limitations have become more specifically known. Obviously, such forcible manipulation, with the tearing of adhesions, may result in some flare up of activity of the arthritic inflammatory process. It must not, therefore, be attempted at the height of activity of the disease if one is to avoid increased destruction of intra articular tissues and ankylosis. However, if more conservative measures tried over a period of weeks or months are ineffective in reducing the degree of deformity, and the arthritis is essentially quiescent, it is not necessary to wait until all activity of the arthritic process has ceased

On the whole, the advantages of manipulation outweigh the risk of the procedure even in the face of some residual activity of the arthritic process. The possibility of temporary activation of the inflammatory process is to be feared less than the danger of firm fibrous ankylosis because of delay in undertaking manipulation. It must be remembered, too, that atrophy progresses as the joints remain immobile. In any event, however, one should anticipate some degree of activation of the process and be prepared to employ such measures as would aid in combating this effect

We frequently give blood transfusions after manipulation, particularly when there is any significant rise in temperature or acceleration of sedimentation rate, and have found them of distinct value. The joints are treated by splinting in the corrected position, and physiotherapy as well as exercises are instituted as early as possible

Manipulation is not entirely free of potential hazards, such as excessive damage to joint structures, rupture of blood vessels, and even fracture of

demineralized bones Experience in the selection of cases the skill of the operator and the gentleness with which manipulation is carried out determine the extent to which damage from this procedure may be avoided

5 Skeletal traction in the line of deformity provides another method of overcoming flexion contracture It is particularly adapted to correction of knee flexion deformities and must sometimes be supplemented by surgical operations directed against shortened tendons of hamstring muscles, the iliotibial band and the posterior portion of the capsule

For very pronounced flexion contractures at the knee when the maximum extension is a right angle operative surgical freeing of the muscular and capsular structures may be combined with skeletal traction to restore normal alignment and a functionally useful knee Surgery would prove likewise effective in long standing contractures which, though not severe present particular resistance to stretching of muscles and capsule and danger of injuring the common peroneal nerve The technique for skeletal traction and the supplementary operative procedures applicable to the correction of arthritic flexion deformities at the knees have been well described and illustrated by Haggart (1935)

6 Surgical procedures aiming at the rehabilitation of arthritic cripples present important aids in the treatment of the most disabling effects of atrophic arthritis The skillful handling of even advanced cases by co-operative efforts of the internist and competent orthopedist may restore some bedridden invalids to lives of relative usefulness and happiness Even partial restoration of joint function to one who has been completely disabled may be worth much more than full restoration of function to one who was only partially incapacitated

The success of surgical treatment of arthritic deformities depends upon many factors (1) the proper selection of patients from the standpoint of quiescence of the arthritic process, (2) the morale of the patient and his understanding of and enthusiasm for the undertaking on which he is embarking (3) the competence of the orthopedist and his experience in the surgery of arthritic joints (4) available facilities for the necessary preoperative and postoperative management of these cases

Quiescence of the arthritic inflammatory process is a most essential requisite otherwise there is danger of recurrence The longer the process has been quiescent the less likely is the possibility of a flare up after operation Determining when an arthritic joint is quiescent or active is not always a simple matter In addition to the clinical evidence from the history and examination one may obtain helpful data from the sedimentation test and blood count particularly from study of the degree of maturation of the polymorphonuclear cells A normal sedimentation rate and the absence of too great a proportion of nonfilamented neutrophils are important con-

firmatory data when all the other evidence points in the direction of quiescence of the inflammatory process. The significance of slight or moderate acceleration of the sedimentation rate, when clinical evidence indicates inactivity of the arthritic process is more difficult to evaluate. The author's experience indicates that acceleration of the sedimentation rate beyond its normal range is probably always a reflection of residual activity of inflammation, definitely so pathologically although not necessarily of sufficient significance clinically. In other words, a joint practically inactive by all clinical criteria may nevertheless be the site of old cartilage erosion and of a thick synovial pannus which harbor inflammatory foci and cause an increased rate of sedimentation. The inflammatory process there may be so attenuated, however, as to offer no drawback to successful surgical reconstruction of the joint. Moreover, with multiple joint involvement, an increased rate of sedimentation may be the result of activity of infection in joints other than those for which operation is contemplated.

For all these reasons it is at times necessary to draw conclusions as to whether an old arthritic joint is ripe for surgical intervention on the basis of clinical evidence alone. Absence of pain and tenderness for a long time (for at least six months or a year), total freedom from even temporary exacerbations of inflammation and soreness—these would indicate quiescence of the inflammatory process. Pain and soreness on motion at the joint may, however, result from mechanical causes—cartilage erosion, slight dislocations of a flail joint, impingement upon tags of synovial pannus—even though the inflammatory process has been entirely extinguished. Experience helps greatly in deciding whether the arthritis is active or quiescent, but withal one may not be quite sure. In that case it is generally better to wait and observe the situation more closely. Sometimes, however, it is advisable to carry out certain operative procedures before complete quiescence has been attained, in order to save a joint threatened with complete loss of function.

The crippled patient who is to be accepted for surgical rehabilitation must have intelligence, a clear idea of what is likely to be accomplished, and an equally clear idea of what cannot be accomplished in his case. He is not a good candidate for an extensive program of rehabilitation if he does not display actual enthusiasm at the prospect of even partial restoration of useful function. As Dr. Osgood put it: "He must beg for these measures, as these patients often will if the proposition is put to them properly."

The orthopedist must be interested in this difficult branch of his surgical practice and have a special experience in the management of these cases and their peculiar problems.

The facilities for adequate preoperative and postoperative care are no less important than all the other attributes of the surgical problem. A perfectly free, well fashioned articulation, anatomically, is of little use

unless there is adequate muscle power for function. Preoperative muscle training is of great help, because, with such preparation, the patient can start the various muscle exercises required soon after operation and thus attain muscular power and control earlier and more effectively. Physical therapy, generally necessary for an extended period of time after operation, really consolidates all its gains.

A variety of operative procedures may be employed in the rehabilitation of crippled patients. I shall mention briefly only the more important ones or those most frequently employed.

Synovectomy, or excision of thickened hyperplastic synovial membrane, may afford relief from a mechanical handicap which cannot be eliminated otherwise. The thickened synovia, thrown into folds and with many projecting villi, causes mechanical interference with joint function or is the cause of pain as the projecting folds are caught and pinched between the articular surfaces of the bones. Such thickened synovia, requiring excision, is encountered most often in the knees. It is understood, of course, that the operation is applicable only to cases of long standing in which the inflammatory process is quiescent or at a standstill. Synovectomy should not be performed before the effect of rest and medical treatment has been noted for, in the earlier stages when the inflammatory process is still active, remarkable resolution of thickening may ensue as the inflammatory process subsides under adequate medical care. Furthermore, indiscriminate resort to this surgical procedure may lead to poor results with limitation of joint motion and recurrence of pain in those who should not have been operated upon. Following operation, in well selected cases, there occurs regeneration of a sufficiently good substitute for the synovial lining to yield a good, freely movable, painless joint. The operation is not formidable. The beginning of active motion may be encouraged within a week after operation, and weight bearing may be possible within four to eight weeks.

Capsulotomy or capsuloplasty may yield complete restoration of joint function which might otherwise be impossible to secure. Capsular contraction alone may cause marked functional impairment even when there is but slight damage to the articular cartilage and synovial membrane. Many years ago Dr. David Silver emphasized the role of the capsule in joint contracture and described a method of freeing the joint from such capsular changes. Wilson, too, later emphasized the value of posterior capsuloplasty in certain flexion contractures of the knee. He pointed out the tremendous handicap which results from limitation of full extension of the knees resulting from contracture of the posterior capsules, particularly since such contracture is frequently bilateral and when pronounced, may lead to complete invalidism. The operation is of course applicable to those cases in which the capsular contracture is of such a degree or duration that it cannot be

readily or safely relieved by such procedures as manual manipulation or wedging of casts. The operation consists of an incision over the lateral aspect of the knee, dividing the iliotibial band of the fascia lata, lengthening of the biceps tendon, opening of the posterior compartment of the knee and stripping away the attachments of the posterior capsular ligaments to the femur, manipulating the knee into complete extension, closure of the incision and the application of a plaster cast to maintain normal alignment. Periods of active motion may be begun at the end of two or three weeks, nightly splinting should be continued for many months to prevent recurrence of the contracture. Walking with the aid of a brace may be permitted at the end of five or six weeks.

Osteotomies may be of use in correcting flexion deformities of the knees when in addition to marked capsular contracture there is also considerable articular damage limiting greatly all motions at the knees. Osteotomies may also correct adducted legs when there is fixation at the hips.

Arthroplasty, the reconstruction of joints or the construction of new ones, may restore useful motion where previously none existed because of fibrous or bony ankylosis. It may restore painless motion when damage to articular cartilage has not led to fusion of the joint, but has caused equally severe incapacity through pain. Wilson summarizes his experience with arthroplasties as follows:

My first operations for the relief of ankylosis in atrophic arthritis were performed approximately fifteen years ago and were accompanied by misgivings because of the warnings of experienced surgeons that such operations were inadvisable in this disease. Tragedies have not resulted, there have been but few complete failures and on the whole the results have exceeded expectations. This experience has tended to make me broaden the indications for its use and I do not hesitate to advise it now in cases which I would not have considered operable by former standards. Even when there is later recurrence of the arthritis the new joint produced by arthroplasty retains its good function and shows no evidence of involvement. The elbow and jaw give the best results, the knee the next best, and the hip follows closely after. Weight bearing after arthroplasty of the knee or hip should be postponed until the end of twelve weeks and then only with brace protection. Arthroplasty of the finger joints is a tedious procedure but worthwhile results can be obtained when it is performed with proper technique.

Multiple arthroplasties have been performed at different times in the same patients with, in some instances, remarkable functional improvement. In case of bilateral ankylosis of the elbows double arthroplasty may be counted upon to give good results. In case of bilateral ankylosis of the knees it is usually sufficient to perform arthroplasty of a single knee although in 2 patients I have done a double arthroplasty with fair results. In case of ankylosis of both hips and both knees the attempt should be made to restore motion in one hip and in the oppo-

site knee. Later it can be decided whether something should be attempted for the second hip.

Even in the most widespread arthritis with multiple ankylosis of joints leading to complete incapacity, multiple arthroplasties have been performed thus enabling the patients to walk and use their arms so that they are no longer dependent on others for their every need.

The arthroplasty technique perfected by Smith-Petersen employing vitallium molds is an ingenious procedure which appears very promising. It is particularly applicable for reconstruction of badly destroyed hip joints in advanced hypertrophic (osteo-) arthritis and of ankylosed hips in burned out cases of atrophic arthritis.

Arthrodesis although conceivably of use on rare occasions should be avoided whenever possible in atrophic arthritis. The general aim of bringing added rather than less motion to joints in this disease will bring greater satisfaction and fewer regrets.

Tenotomy is relatively limited in its usefulness in the surgical treatment of arthritic deformities. Subcutaneous tenotomies are sometimes useful however in the correction of certain deformities of the ankles or toes. As already pointed out, lengthening of the biceps tendon is sometimes required to permit perfect extension of the knee when correcting long standing contractures of unusually pronounced degree.

III PREVENTION AND CORRECTION OF DEFORMITIES IN THE FEET

The feet seldom escape arthritic involvement when the condition involves multiple joints. Arthritic affections of the feet constitute a serious problem because they disturb the integrity of the pedestal for the entire body. Even normal feet are subject to attacks of arthritis but when it is realized how frequently various types of static abnormality in the feet exist in apparently healthy individuals it is not surprising that the feet are affected so often and that the condition has a tendency to stubborn progression unless circumstances are made most propitious for recovery. The feet present a particularly pressing demand for relief because the pain is likely to be severe and may be incapacitating to an extreme.

The trauma of walking and weight bearing is more than we can fairly expect an actively inflamed arthritic foot to bear. The intense pain is caused not only by direct effects of inflammation but also by secondary effects of ligamentous and muscular strain and of abnormal pressure at points of deformity. The debility which generally predisposes to the onset of generalized arthritis seldom fails to level its effects on the supporting

structures of the feet. Frequently such physical debility only accentuates a static foot deformity which antedated by a long time the final episode of physical exhaustion, and, in some cases, actually contributed to its appearance. In any event, normal foot posture in a patient with arthritis is rare, evidence of foot strain most common.

That the inflammatory process in the feet subsides in some cases despite the burden of postural foot strain is attributable to the inherent tendency of the disease toward cure. To put every patient to such a test, in the face of static defects which cry for correction, is to sentence most of them to unnecessary torture and to jeopardize their chances for full recovery. The strain of the whole body weight, the trauma of walking, the protective devices the patient unconsciously brings into play to alleviate pain—all these conspire to produce deformities, which interfere with the smooth functioning of the feet. Nor is the effect confined there, for it may be a source of postural strain to the knees, hips, and back, severe enough to maintain the activity of arthritis in those joints. The author has encountered cases in which correction of badly pronated feet and flattened arches opened the way for ultimate cure of the arthritis there, as well as in the knees, previously refractory to all other treatment.

As in all other phases of treatment, prevention or early correction of deformities is most important. Neglect of such early care brings about a train of increasingly pronounced and fixed deformities which are exceedingly difficult to manage. It leads to cartilage destruction, opening the way to ankylosis and loss of flexibility of the foot, or to muscle spasm and strain, producing serious static defects, which may become fixed, through contracture of muscles and ligaments. Owing to the complicated structure of the foot and the interdependence of its various functional units, every ill effect produced is reflected in secondary changes in adjacent structures, with constantly increasing damage. If deformities are permitted to develop, they subject parts of the foot to undue pressure, leading, in turn, to exquisitely painful callosities.

There is no more important measure of early treatment in the acute, subacute, or active chronic stages than absolute rest in bed. Weight bearing must be entirely eliminated. The feet must be supported in a neutral position to prevent deformity, and a cradle should be provided over them to eliminate pressure of bedclothes. To eliminate the effect of muscle spasm, plaster or "castex" boots, or metal splints may be employed. These boots are bivalved, made easily removable for the application of physical therapy.

Active exercises aimed at maintaining or improving muscle tone and foot posture should be instituted as early as possible, their range and frequency being increased as the subsidence of activity of the inflammatory process permits. Only when all signs of activity of the arthritic process in the feet

have disappeared should the patient be permitted to begin walking. Prior to this full correction should have been made of all residual static defects. Shoes should be correctly fitted and specifically altered to compensate for distortion of the arches, pronation of the feet, or whatever other defect may exist. A variety of mechanical aids (illustrated in the next chapter) may be utilized to prevent deformity and relieve strain and pain on walking.

Developed deformities may be corrected by gentle manipulation and physiotherapy, supplemented when indicated by the application of plaster casts to maintain the correction secured. Always the aim should be to attain as good a weight bearing position as possible. More severe deformities may require manipulation under anesthesia followed by supportive treatment. The requisite principles for successful manipulation have already been discussed.

Surgical correction of even severe and extremely disabling deformities may restore sufficient use of the feet to convert a bedridden invalid into a useful person. Surgical treatment of advanced foot deformities may constitute a long drawn-out, difficult but not insurmountable task, the results of which may be more gratifying than could be imagined from a view of the foot before operation.

These in general are some of the corrective procedures which may be resorted to in the rehabilitation of patients who have for one reason or another drifted to the stage of joint deformity and physical incapacity. The author has not attempted to catalogue all of the many corrective measures which are actually called upon in the treatment of this phase of arthritis. To do so would only begot the statement of the principles involved; these it is desired to emphasize here. In actual practice the store of therapeutic measures available for the correction of deformity will be as extensive or as limited as the resourcefulness of the physician-orthopedist group in charge.

SOME AXIOMS IN THE TREATMENT OF ARTHRITIS WITH REFERENCE TO PREVENTION OF DEFORMITIES

- 1 Do not run away from a crippled arthritic. A deformed patient is not necessarily a hopeless one.
- 2 All the effort required to prevent joint deformity is infinitesimal in proportion to that required for its correction after it has developed.
- 3 Treat the joints locally while you treat the patient systemically.
- 4 Anticipate and prevent muscle spasm—nature's attempt to splint the joints—by employing external support.
- 5 Always combine rest with exercise of the joints. The more acute the

- process the greater the need for rest, but never to the point of total exclusion of exercise
- 6 Passive or active motion once or twice a day, during the most acute phases of the disease, will insure against ankylosis in most cases
 - 7 Driving the patient to excessive use of the joints, particularly when the arthritic process is active, will do more harm than good
 - 8 When an arthritic patient, treated by a properly balanced ratio of rest and exercise, develops ankylosis of joints, while in bed, it may be accepted that such deformity occurred despite the bed rest and not be cause of it
 - 9 Most deformities and limitation of mobility of joints, are caused not by bony ankylosis, but by fibrous contracture of periarthritic soft tissues and muscles
 - 10 Partial, but useful, restoration of function to an individual with arthritis who has been totally disabled is more gratifying an achievement than complete restoration to normal of an individual who was only partially incapacitated

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[For a list of books and monographs dealing with the general aspects of
 chronic rheumatic disease (including considerations on the subject of the
 present chapter) see page 527]

CHAPTER XV

AN ILLUSTRATED GUIDE TO THE RECOGNITION OF THE CHARACTER AND CAUSES OF ARTHRITIC DEFORMITIES AND OF THE MEANS FOR THEIR PREVENTION AND CORRECTION

THE HEAD

Character of deformity generally encountered

- 1 Forward flexion and rotation of the cervical spine (Fig 30)

Its causes

- 1 Spasm of muscles of the neck, induced by arthritic involvement of the cervical spine, tends to rotate or flex the head in the direction of strongest muscle pull
- 2 Fixation of the neck in an abnormal position by muscle spasm, affords some relief from discomfort, hence, the patient maintains that position
- 3 The head supported by a large pillow increases the tendency to forward flexion deformity (Fig 31)
- 4 Adaptive shortening of the spastic muscles ensues
- 5 Finally, stiffness and contracture of the spinal ligaments or actual ankylosis of the cervical spine perpetuates the deformity

To prevent it

- 1 Do not support head with a large pillow during recumbency, use a small one or none (Fig 32)
- 2 Correct the position of the neck to normal, with the face looking straight forward, if abnormal pull of the head occurs in any direction Splint with a rigid cervical collar, or with sand bags, or by traction on a head sling (Figs 33, 34)
- 3 Employ light massage and exercises to prevent ankylosis
- 4 If ankylosis appears inevitable, apply a cervical collar which will maintain the erect position of the head, with the face looking directly forward

for correction of deformity, if already present

Note Correction of deformity in the cervical spine is frequently altogether impossible, its prevention may generally be successfully achieved



FIG. 30 Fixed forward flexion of the head in a patient with atrophic arthritis. Note also the marked flexion deformity of the dorsal spine, with kyphosis which masks, to an extent the full degree of deformity in the cervical region. Preventive measures to maintain extension of the cervical and dorsal spine were never instituted in the early, formative stages of these deformities.

1. If caused by muscle spasm or contracture of muscles, traction on a head sling may, in time, restore the normal position of the head
2. If true ankylosis has not occurred, physiotherapy, supplemented by manual stretching and the application of plaster collars, each maintaining the slowly progressive correction of the deformity, may ultimately yield considerable improvement.



FIG 31 The use of several pillows to support the head flexing the cervical spine leading thus to flexion deformity of the head



FIG 32 The patient lying in bed without a pillow, with the cervical spine in extension to prevent deformity of the head



FIG. 35 A cervical collar made of felt such as is used to maintain the normal position of the cervical spine and head. This collar can be easily removed for the application of physical therapy.

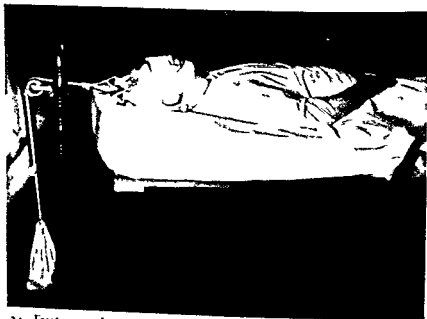


FIG. 34 Traction such as is employed to prevent or correct flexion deformity at the

THE THORACIC SPINE

Character of deformity generally encountered

- 1 Forward flexion (Fig 35)
- 2 Drooping and fixation of the ribs flattening and fixation of the chest



FIG 35 Atrophic arthritis of the spine (Marie Strumpell spondylitis) showing forward bowing at the dorsal spine with rigid fixation in flexion the result of calcification of the spinal ligaments

in the position of expiration when the posterior articulations of the ribs are involved

- 3 Interference with expansion of the chest and inspiration impossible

Its causes

- 1 The patient assumes an abnormal position in bed with the spine flexed and supported by many pillows (Fig 36)
- 2 Pain on motion and stiffness in the spine induces limitation of full extension thus lessening the discomfort of activity
- 3 Gradual contracture of the anterior spinal ligaments as they become

infiltrated with calcium leads to rigidity of the spine in the abnormal position

- 4 Deformity is accentuated by atrophy of paraspinal extensor muscles



FIG. 36 The position in bed favoring deformity of the spine. Note the forward bowing of the spine and incidentally the adducted arms, flexed elbows and wrists, the ulnar deviation of the fingers.

overbalanced by less affected abdominal flexor muscles plus effect of gravity in the erect position.

To prevent it

- 1 Institute rest in bed in supervised recumbent position for weeks or months at the earliest signs of arthritic involvement in the spine (Fig 37)
- 2 Exercise every precaution to maintain the normal contour of the spine
- 3 See that the bed is firm and prevent it from sagging by the use of a firm mattress and a board between it and the spring
- 4 Induce hyperextension of the spine and full extension of the chest cage by placing a pillow under the dorsal spine and bringing the hands under the head for short periods of about one half hour several times through the day (Fig 38)
- 5 Apply heat and massage for reducing muscle spasm and pain
- 6 When the patient is improved institute active postural exercises aimed particularly at development of the back and abdominal muscles as well as the attainment of a normal gait and foot alignment to aid in the prevention of recurrence

- 7 When the patient becomes ambulatory provide him with an appropriate back brace to aid in the maintenance of the normal position of the spine (Fig 39)



FIG 37 The position of the patient in bed with the dorsal and cervical spine in extension preventing flexion deformity



FIG 38 To prevent forward bowing of the dorsal spine and to induce maximum expansion of the chest the patient rests for periods of twenty to thirty minutes several times a day on a pillow placed under the dorsal spine and the hands under the head

- 8 If ankylosis is inevitable allow it to occur with the spine in full extension and the chest in expansion

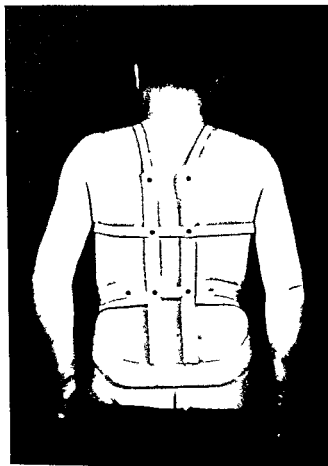


FIG. 39 A back brace designed to maintain the normal position of the spine while the patient is ambulatory after preliminary rest in hyperextension has been carried out

For correction of deformity, if already present

Note Correction of flexion deformity of the spine, pathetically disabling and disfiguring is frequently altogether impossible, its prevention may generally be successfully attained

- 1 Attempt hyperextension of the spine with the patient at rest in bed. Allow no head pillows. Later a pillow under the spine, or a sandbag or a plaster hyperextension frame may be employed
- 2 Physiotherapy may facilitate the process of hyperextension, by relieving muscle spasm and discomfort
- 3 Postural exercises should be employed
- 4 A back brace should be fitted to maintain the correction attained

THE LUMBAR SPINE



FIG. 40 A plaster jacket designed to immobilize the lumbar spine and relax muscle spasm



FIG. 41 A lumbosacral pad employed to maintain the *normal* lordosis at the lumbar spine. Note the non sagging bed, with the board between the mattress and spring

THE SHOULDER



FIG. 4. Limitation of abduction of arms in a case of atrophic arthritis resulting from long continued maintenance of arms in a position of abduction and internal rotation.



FIG. 43. Maximum abduction of arm possible when scapular motion is eliminated.

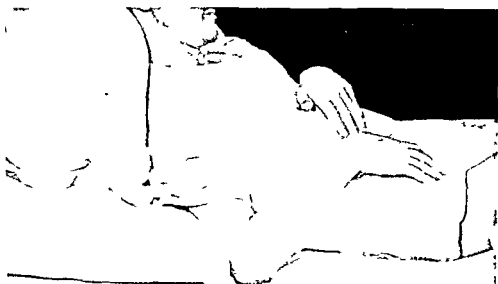


FIG 44 The position, in bed, favoring adduction deformity at the shoulders. Note, incidentally, flexion of the spine elbows and wrists and ulnar deviation of the fingers

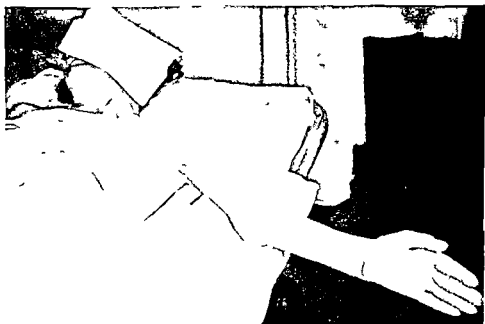


FIG 45 Adduction deformity, atrophy of muscles about the shoulder and contracture of pectoralis major muscle, with resulting limitation of full abduction of arm in a case of atrophic arthritis

Character of deformity generally encountered

- 1 Fixation of arms in adduction and internal rotation (Figs 42, 43)



FIG 46 Traction employed to correct adduction deformity at shoulder Flexion deformity at the wrist and fingers is prevented by a plaster cock up splint with a forward extension to the fingers

Its causes

- 1 Painful joint motion and sore muscles encourage total inactivity
- 2 The position of the arms, closely adducted to the body and internally rotated, aggravates the deformity (Fig 44)
- 3 Subsequently, atrophy of abductor group of muscles and contracture of capsule occur
- 4 The pectoralis major, subscapularis, latissimus dorsi and teres major muscles undergo contracture (Fig 45)



FIG 47 Position of abduction and external rotation of the arms assumed at intervals during the day to prevent the development of a frozen shoulder in adduction and internal rotation

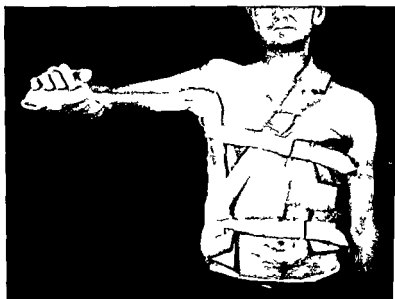


FIG 48 An aeroplane splint employed to prevent adduction deformity at the shoulder joint. It is easily removed for the application of heat, massage and exercises

To prevent it

1. Maintain the arm in a position of abduction and full external rotation by means of sand bags, balanced traction, or placement of hands under the head for short periods several times each day. Ambulatory patients can maintain the desired position by means of an aeroplane splint (Figs 46, 47, 48).
2. Employ baking, massage and exercise until shoulder is entirely recovered.
3. Institute a program of occupational therapy.

For correction of deformity if already present

1. Employ traction and exercises.
2. When marked atrophy is present, induce faradic stimulation of muscles followed by passive then active exercises.
3. When contracture and adhesions in capsule exist, manipulate shoulder under anesthesia. Then maintain corrected position by traction or abduction splint, physiotherapy and exercises.

THE ELBOW

Character of deformity generally encountered

1. Flexion at elbow and pronation of forearm (Figs 49, 50).

Its causes

1. Pain occurs on motion at the elbow.
2. The elbow is held in position of flexion with pronation of the hand (Fig 51).
3. This position is maintained day and night because it affords comfort and because it is natural even with normal muscle balance.

To prevent it

1. Maintain full extension at elbow by means of a splint put on for the night (Fig 52).
2. Encourage use of elbow at intervals during the day by passive or active exercises after baking and massage.
3. Encourage full use of elbow during the day as improvement occurs and splint in full extension for the night until recovery.

For correction of deformity if already present

1. Force extension or flexion at elbow by a series of splints, each securing a little more flexion or extension and supination as desired.
2. Straighten elbow by wedging of a plaster cast. Turnbuckles or Turner irons may also be used (Fig 53).



FIG. 49 Flexion deformity at elbow and pronation of forearm in a case of atrophic arthritis showing maximum degrees of extension and flexion possible in this case and revealing incidentally the flexed arm and pronated hand lying across the body—the position generally maintained by the patient in bed eventually producing the flexion deformity.

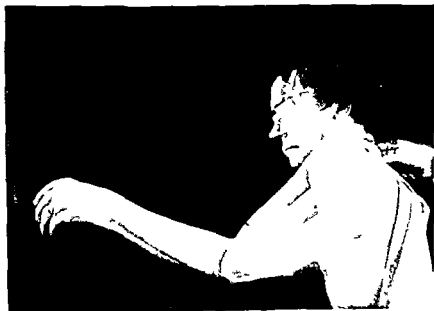


FIG. 50 Flexion deformity at elbow and pronation of forearm in a case of atrophic arthritis showing maximum degrees of extension and flexion possible in this case.



FIG 51 A bad position in bed, favoring flexion deformity at the elbows. Note incidentally, the flexed spine, adduction and internal rotation of the arms, flexed wrists and ulnar deviation of the fingers.

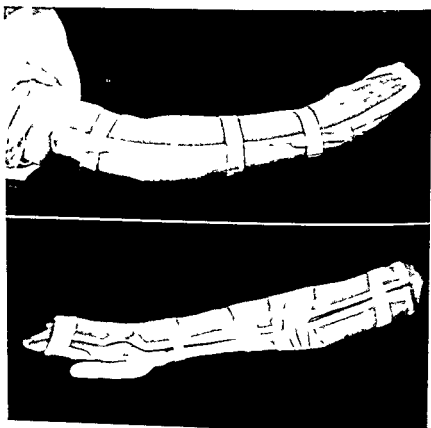


FIG 52 A plaster splint above, and below, one made of a new cellulose compound material (castex) employed to prevent flexion deformities at the elbow, wrist and fingers, in atrophic arthritis. The elbow illustrated in the upper section presents slight flexion deformity and is temporarily splinted in that position. As spasm of the flexor muscles of the elbow disappears with immobilization and physiotherapy, and the arm straightens, a new splint will be made to maintain the corrected position. These splints are removable, permitting the application of heat, massage and exercises.

Note Long standing flexion deformities at the elbow associated with shortening of the flexor tendons may be straightened by slow wedging of casts, but restitution of the full range of motion is a slow, painful



FIG 53 A plaster cast cut at the elbow and wedged—a means employed to correct flexion deformity at the elbow

procedure usually requiring a long time. There is also a tendency for the contracture to recur. Therefore, we do not advocate straightening of slightly flexed elbows, particularly if there is no limitation in the range of flexion.

- 3 When the infectious process is totally inactive perform arthroplasty on elbow, an operation which is generally successful.

THE WRIST

Character of deformity generally encountered

- 1 Palmar flexion and pronation at the wrist and ulnar deviation of the fingers (Figs 54 55)

Its causes

- 1 Spasm of the stronger set of flexors of the wrist, aided by gravity, pull the wrist into a position of flexion.
- 2 The position of the hand, generally resting on the body, with the elbow and wrist flexed leads to pronation of the hand and tends toward ulnar deviation of the fingers (Fig 56)



FIG. 54. Extreme degree of muscle atrophy and palmar flexion deformity at the wrists caused by spasm and contracture of the flexor muscles in a case of atrophic arthritis. This deformity could have been prevented by means of simple cock up splints applied at the earliest signs of involvement of the wrists.



FIG. 55. Palmar flexion deformity and subluxation at the left wrist and slight ulnar deviation of the fingers in a case of long standing atrophic arthritis.

- 3 Pain in the wrist soreness and weakness of the muscles and later atrophy of muscles discourage extension
- 4 Shortening of the flexors of the wrist and contracture of the capsule of



FIG. 56 A bad position of the arms favoring the development of flexion deformities at the wrists and ulnar deviation of the fingers

the joint as well as cartilage destruction and fibrous or bony ankylosis maintain the deformity

To prevent it

- 1 Maintain the wrist in about 30 degrees of dorsiflexion by a cock up splint made of plaster of paris or metal or castex (Figs 57 58 59)
- 2 Leave splint on all night and during most of the day in acute cases. But remove splint for periods during the day carrying out baking massage passive and active exercises at this time
- 3 Exercise through the fullest arc of painless motion once or twice a day. This will generally insure against ankylosis
- 4 As the activity of the process subsides the splints may be used only at night

For correction of deformity if already present

- 1 Induce gradual extension (dorsiflexion) of the wrist by means of a series of plaster cock up splints each aiming for a bit more extension than the previous one
- 2 Manipulate the wrist under anesthesia when the activity of the arthritic process is quiescent or burnt out and follow by splinting in the corrected position heat massage and exercises
- 3 As a last resort surgical ankylosis of the wrist in a position of about 30

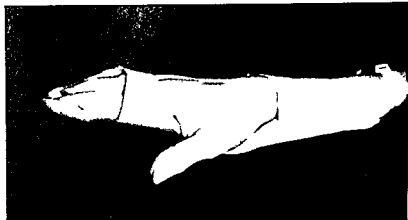


FIG. 57 A light plaster cock up splint designed to prevent flexion deformity at the wrist with an extension under the hand to prevent flexion deformity of the fingers. This splint is easily removed for the application of heat, massage and active exercises.



FIG. 58 A simple light metal cock up splint used to prevent the tendency to flexion deformity at the wrist.

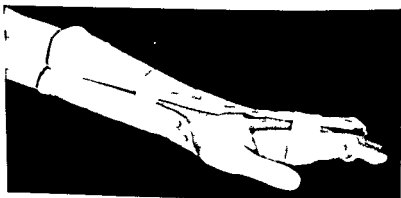


FIG. 59 A cast made of a new cellulose compound bandage material (castex) used to maintain extension of the wrist and fingers. The cast is cut along one side and may be removed easily for the application of physical therapy. These casts are light, durable and waterproof. Ideally suited for splinting of arthritic joints.

degrees of dorsiflexion yields a painless joint in the most useful position

THE HAND AND FINGERS

Character of deformity generally encountered

- 1 Flat hand'—flattening of the palmar arch
- 2 Subluxation of the thumb is frequent
- 3 Ulnar deviation of the fingers
- 4 Contracture at the metacarpophalangeal joints in hyperextension or flexion
- 5 Flexion deformities usually, sometimes hyperextension deformities, at the interphalangeal joints (Figs 60, 61, 62)
- 6 Dislocations at the interphalangeal or metacarpophalangeal joints

Its causes

- 1 Atrophy of the small muscles of the hands and the palms bearing most of the body weight, as the patient attempts to rise and sit down, lead to flattening of the palmar arch
- 2 The position in which the patient generally rests the hand, abetted by spasm and later contracture of the flexor muscles of the fingers and capsules of the smaller joints produces deformity of the fingers (Fig 63)

To prevent it

- 1 Splint the palmar arch and fingers in their normal position by a properly molded forward extension of a cock up wrist splint (Fig 64)
- 2 Introduce physiotherapy and exercises during the day
- 3 As soon as the arthritic process has begun to subside, institute occupational therapy

For correction of deformity, if already present

- 1 Promote traction by means of a banjo splint (Fig 65)
- 2 Gradually stretch the contracted capsules and tendons by means of plaster of paris or metal splints
- 3 Manipulate the fingers and follow by splinting in the corrected position, supplemented by massage, exercises and occupational therapy
- 4 Release capsular contracture surgically as is occasionally necessary

Note If much atrophy of the extensor muscles of the fingers exists in association with long standing flexion deformities straightening of the fingers may result in a better looking hand, but one that is less useful functionally

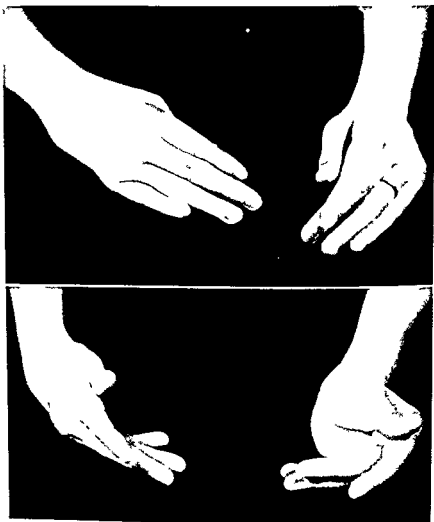


FIG. 60. Marked contracture, with flexion deformity at the metacarpophalangeal joints in a case of chronic atrophic arthritis.



FIG. 61 Moderate degree of ulnar deviation of the fingers of the right hand hyper extension deformity at the proximal phalangeal joints of the middle fingers of both hands periarticular swelling atrophy of smaller muscles and limitation of full extent of flexion of fingers in chronic atrophic arthritis



FIG. 62 Marked flexion and hyperextension deformities with ankylosis of the fingers, in a case of advanced atrophic arthritis in which practically every joint has been affected. Note also the glossy taut skin which has a distinctly sclerodermatous character over the distal parts of the hands, the scleroderma having developed apparently long after the arthritis.



FIG. 63 The position of the hands crossed over the chest leading to flexion of the wrists as well as ulnar deviation of the fingers.



FIG. 64 Cock up splint extended forward under the hand to maintain the fingers in neutral extension and to prevent ulnar deviation of the hand. The splint is removed during the day for the application of heat, massage and exercises.



FIG 65 Marked flexion deformities and ulnar deviation of the fingers in a case of chronic atrophic arthritis undergoing correction by manipulation under anesthesia followed by the application of banjo splints

THE HIP

Character of deformity generally encountered

- 1 Flexion and contracture
- 2 Adduction of the leg (Fig 66)



FIG 66 An arthritic deformed with flexion and adduction deformities at the hips flexion deformities at the knees and slight equinus at the feet with ankylosis in the positions indicated in the photograph in a case of widespread atrophic arthritis. Note the glossy thin skin of scleroderma extending from the knees to the peripheral extremities the sclerodermatous change having developed apparently long after the onset of the arthritis. The scar over the right hip region resulted from healing of an old decubitus ulcer.

Its causes

- 1 The position of the leg is assumed because of pain from which relief is sought by flexion of the thigh. Even in health it is a common position of relaxation (Fig 67)
- 2 This position is maintained by muscle spasm later atrophy and shortening of muscles or ankylosis.

To prevent it

- 1 Keep patient at rest in bed when there is evidence of arthritic involvement of the hip relieving the joint of the strain of weight bearing
- 2 Maintain position of extension by traction if necessary

- 3 If owing to severe pain muscle spasm is extremely marked it may be reduced by traction and extension and maintained by continuous balanced traction. Rest may also be induced by means of a plaster spica.

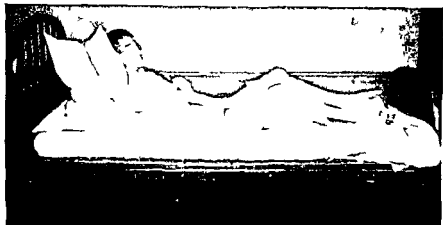


FIG. 6 The position generally assumed in bed with flexion and adduction of the hips leading to the characteristic flexion and adduction deformity.

cut into an anterior and posterior shell which permits removal for the application of physical therapy and exercises.

- 4 Do not keep the hip completely immobilized for long periods, that invites ankylosis. Remove the weights or splint daily for periods of baking and exercises to prevent fixation.
- 5 Eliminate weight bearing until the arthritic process in the hip has subsided and all muscle spasm has disappeared.

For correction of deformity, if already present

- 1 Introduce traction in the line of deformity continued over an extended period of time.
- 2 Manipulate under anesthesia followed by traction or immobilization in a cast for several days with subsequent application of physical therapy including massage and exercises and splinting or traction to prevent recurrence of deformity. Manipulation should not be attempted when there is evidence of much activity of the arthritic process in the hip.
- 3 Consider arthroplasty if there is ankylosis and the disease process is entirely inactive. The Smith-Petersen arthroplasty recently devised employing vitallium molds is an ingenious procedure by means of which ankylosed hips previously hopeless may perhaps be restored to functional usefulness.

THE KNEE

Character of deformity generally encountered

- 1 Flexion deformity, with limitation of full extension and, later, limita

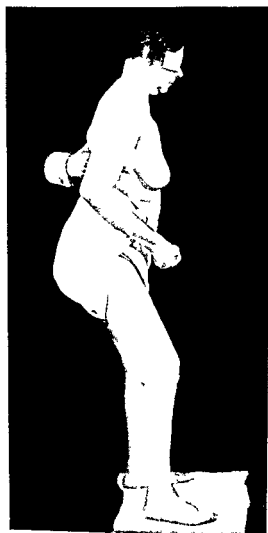


FIG. 65. Multiform postural defects resulting from flexion deformities at the knees and slight pronation at the feet in a case of chronic atrophic arthritis.

- tion of full range of flexion (Fig. 69)
- 2 Backward subluxation of the tibia on the femur
- 3 Outward rotation of the lower leg on the femur (Fig. 70)

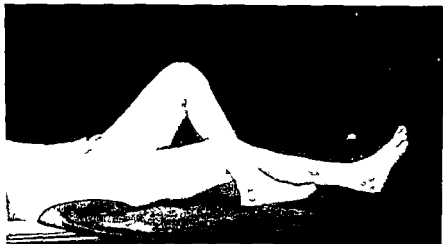


FIG 69 Flexion deformity of the knee with limitation of extension and of full degree of flexion caused by hamstring and capsular contracture in a case of chronic atrophic arthritis



FIG 70 Outward rotation of the lower leg on the femur in case of long standing atrophic arthritis

Its causes

- 1 Pain swelling and soreness of muscles give rise to muscle spasm
- 2 The stronger hamstring muscles pull the knee into flexion



FIG. 71 Flexion deformity at the knees caused by spasm and contracture of the hamstrings in a case of subacute atrophic arthritis the deformity being perpetuated by the pernicious practice of supporting the bent knees with pillows. Note also the outward rotation of the leg on the femur and the position of the feet in plantar flexion which may result in a rigid equinus deformity from contracture of the gastrocnemius muscle.

- 3 The patient finds this position relieves discomfort, so he places pillows under the bend of the knee to support it in this abnormal position (Fig. 71)
- 4 The spastic hamstrings become shortened
- 5 Attempts by the patient to straighten the leg become, then progressively more difficult and more painful
- 6 Atrophy of the quadriceps muscle (the extensor of the leg) develops rapidly. Before long the atrophic weak quadriceps is at a total disadvantage in overcoming the spastic or actually shortened hamstrings
- 7 The knee maintained in a position of flexion suffers additionally from contracture of the capsule of the joint particularly in its posterior portion
- 8 Cartilage thinning and later, its complete destruction, opens the way to fibrous ankylosis by proliferation and fibrosis of the synovial pannus. Ultimately bony fusion may supervene
- 9 External rotation of leg is caused by maintained eversion

Note In atrophic arthritis fibrous or bony ankylosis is actually far less frequently the cause of a fixed knee in flexion than are shortening of the hamstrings and capsular contracture

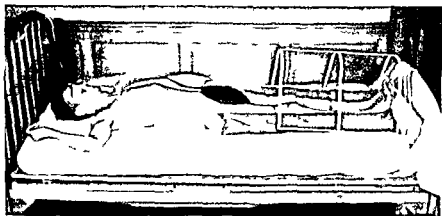


FIG. 72 The patient in bed with the knees straight the legs supported by sandbags at the sides to prevent eversion. Posterior knee splints were unnecessary at this stage because of complete disappearance of spasm of the hamstring as a result of previous splinting. The patient is now free to exercise the limbs to restore the normal tone in the quadriceps muscles.



FIG. 73 A light plaster shell designed to prevent flexion deformity at the knee and plantar flexion (equinus) of the foot in a case of active atrophic arthritis. This splint is easily removed for the application of heat, massage and active exercises.

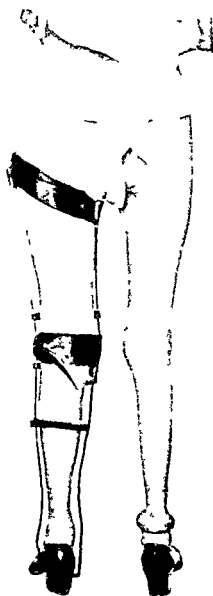


FIG. 74 A supportive brace to lessen the strain of weight bearing at the knee when the patient begins to walk after recovery from arthritis of the knee (From G. H. Hazzart *Surgical Clinics of North America* 15:152-1935)

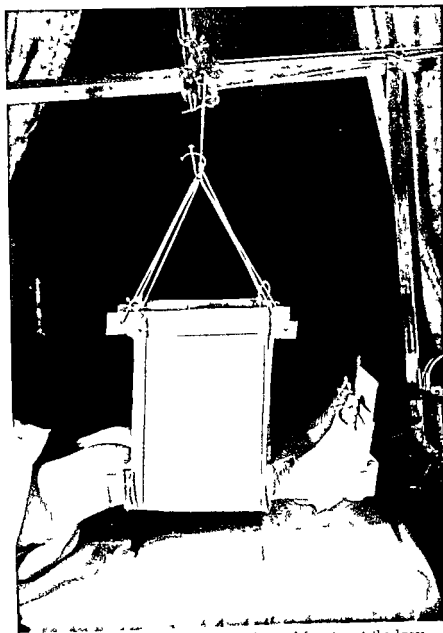


FIG 75 Traction employed to correct flexion deformities at the knees

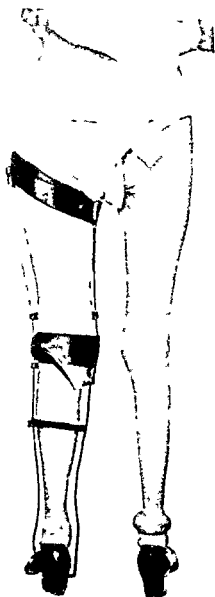


FIG. 74 A supportive brace to lessen the strain of weight bearing at the knee, when the patient begins to walk after recovery from arthritis of the knee (From G. H. Haggart, *Surgical Clinics of North America* 15:1527, 1935)

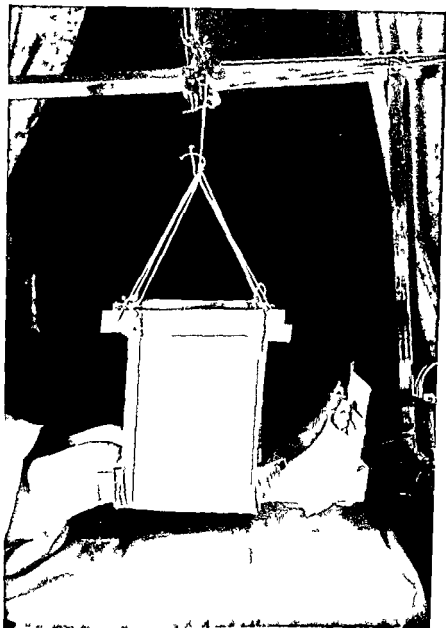


FIG 75 Traction employed to correct flexion deformities at the knees

To prevent it

- 1 At the earliest evidence of arthritic involvement of the knee, eliminate the trauma of weight bearing. The patient should be in bed (Fig 72)

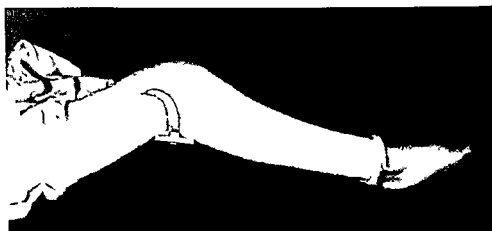


FIG 76 A plaster cast cut under the knee and wedged—a means of correcting flexion deformity at the knee

- 2 Support the knee most of the time but at least during the night by a posterior plaster shell extending from the upper thigh to the toes. This will relieve muscle spasm and pain (Fig 73)
- 3 Introduce physiotherapy, including massage, passive exercises, at first and, later, active motion during the daytime
- 4 As the activity of the arthritic process subsides, employ more active exercise and proportionately less splinting during the day
- 5 Do not allow the patient to bear weight until the activity of the process is entirely gone. If walking is to be attempted soon after quiescence of the arthritis has been achieved, provide a supportive brace or bivalved light plaster cast to lessen the strain of weight bearing (Fig 74)

For correction of deformity, if already present

- 1 Traction in the line of deformity may be carried out (Fig 75)
- 2 Manipulation under anesthesia may be employed when there is only little activity of the arthritic process and slight contracture of the hamstrings and capsule. Manipulation to be followed by traction or splinting, physical therapy and exercises
- 3 Casts may be applied which are cut posteriorly and wedged slowly, thus attaining gradual extension of the knees. The danger of posterior tibial subluxation and peroneal nerve and popliteal artery injury must be considered (Fig 76)

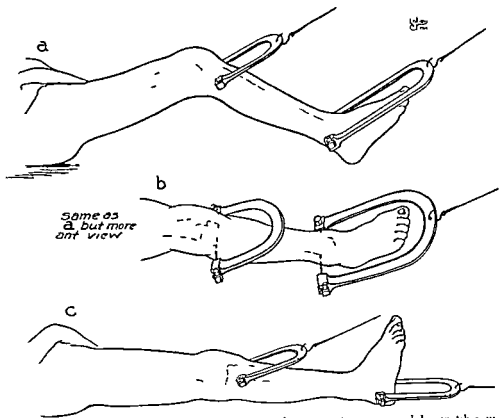


FIG 77 Illustrating the application of skeletal traction to upper and lower tibia in severe knee flexion deformity. Note that the bow holding the lower wire is sufficiently large to permit passage through it of the foot as the knee joint progressively extends. When knee joint extension reaches 165 to 170 degrees the tibial wires are removed and complete correction of the flexion contracture obtained by skeletal traction through os calcis (From G H Haggart *Surgical Clinics of North America* 15 157 1935)

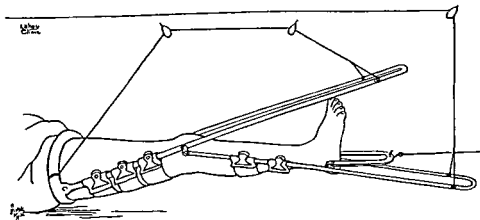


FIG 8 Lower extremity suspended in Thomas Splint with Pierson knee hinge attachment. Final correction of knee joint contracture by skeletal traction at os calcis (From G H Haggart *Surgical Clinics of North America* 15 1527 1935)

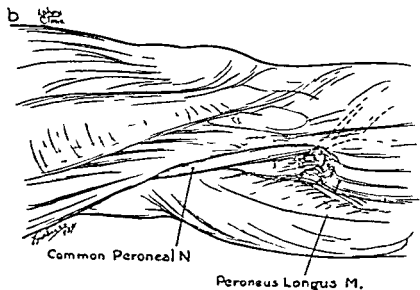
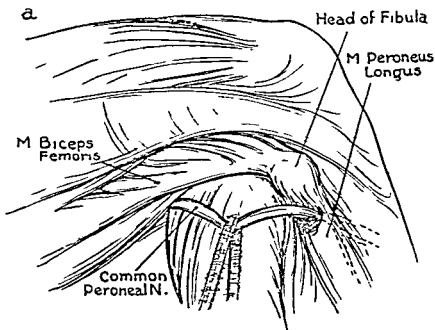


FIG. 73. a Dissection and freeing up common peroneal nerve followed by b partial transverse division of fascia and peroneus longus muscle to permit forward displacement of nerve trunk on complete extension of knee joint. Constriction or stretching injury of the nerve is thus prevented. (From G. H. Huggart *Surgical Clinics of North America* 15:150-1935.)

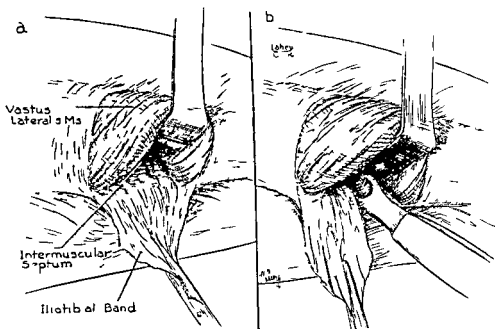


FIG 80 a, Excision of segment of the iliotibial band b Stripping from femur the corresponding portion of lateral intermuscular septum which is continuous with posterior margin of iliotibial band (From G H Haggart *Surgical Clinics of North America* 15 1527 1935)

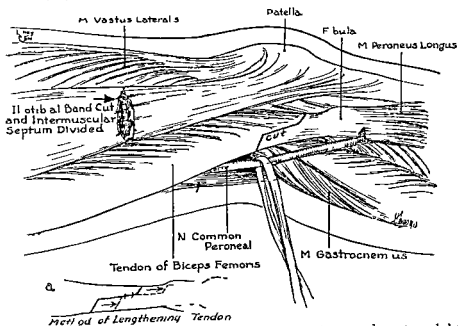


FIG 81 Complete illustration of various steps in operation supplementing skeletal traction for correction of knee flexion contracture (From G H Haggart, *Surgical Clinics of North America* 15 1527 1935)

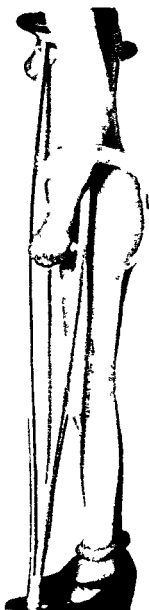
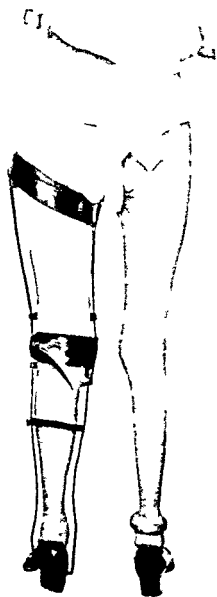


FIG. 5. Patient standing with and without walking caliper brace. Latter is worn and crutch ex a e utilized until leg musculature is well developed. (From G. H. Huggart, *Surgical Cases of North America*, 15, 15 - 1935.)

- 4 Surgical freeing of the periarticular structures combined with skeletal traction may be performed in severe knee flexion deformities. The operation described by Haggart includes division of the lower

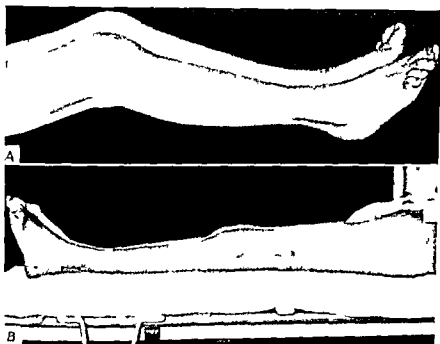


FIG. 83 A Photograph on admission of marked flexion contracture. Patient unable to stand or walk for two years. Note pronounced muscle atrophy. The position shown is the maximum degree of knee extension. B Four weeks following skeletal traction on both lower extremities and open operation on left knee. Note full knee joint extension. (From G. H. Haggart, *Surgical Clinics of North America*, 15, 1527, 1935.)

end of the iliotibial band and of the lateral intermuscular septum; lengthening of the biceps tendon followed by skeletal traction with Kirschner wires. Horizontal division of the peroneus longus muscle permitting forward displacement of the nerve eliminates the danger of constriction or stretching of the common peroneal nerve (Figs 77, 78, 79, 80, 81, 82, 83). (G. H. Haggart, *Surg. Clin. N. Amer.*, 15, 1527, 1935.)

- 5 In the absence of fibrous or bony ankylosis a knee with pronounced and long standing flexion deformity caused by contracture of the posterior capsule may be restored practically to normality by capsulotomy.
- 6 Combined synovectomy and capsulotomy are necessary when marked thickening of the synovia exists in addition to capsular contracture,

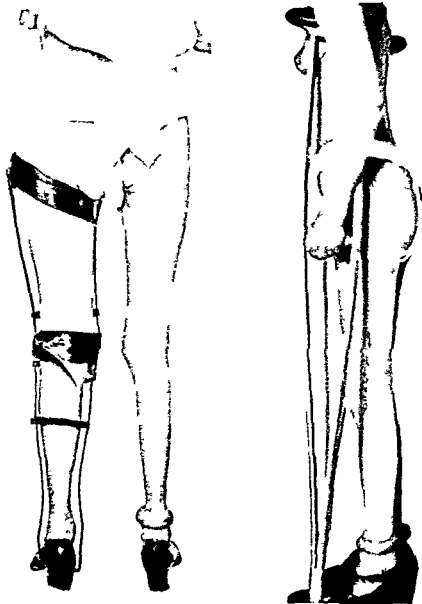


FIG. 5. Patient standing with and without walking caliper brace. Latter is worn and crutches are utilized until leg musculature is well developed. (From C. H. Haggart, *Surgical Clinics of North America*, 15, 1, p. 193, 1932.)

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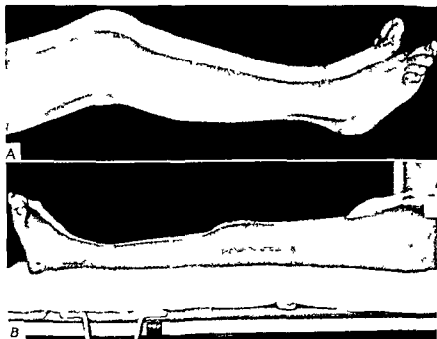


FIG 83 A Photograph on admission of marked flexion contracture. Patient unable to stand or walk for two years. Note pronounced muscle atrophy. The position shown is the maximum degree of knee extension. B Four weeks following skeletal traction on both lower extremities and open operation on left knee. Note full knee joint extension. (From G. H. Haggart *Surgical Clinics of North America* 15, 1527, 1935.)

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- 6 Combined synovectomy and capsulotomy are necessary when marked thickening of the synovia exists in addition to capsular contracture.

but the arthritic process must be inactive if good function is to be maintained

- When there is bony ankylosis arthroplasty of the knee may be performed if there has been a long period of complete inactivity of the arthritic process

THE FOOT

Character of deformity generally encountered

- 1 Pronation and later rigid valgus (Figs 84 85)
- 2 Flattening of the longitudinal arch
- 3 Equinus from neglect of support of foot with resulting contracture of gastrocnemius (Fig 86)
- 4 Flattening or plantar projection at the anterior (metatarsal) arch with formation of hammer toes (Fig 87)

Its causes

- 1 Pronation of the foot may precede the arthritis and may actually give rise to localization of the arthritis there
- 2 Weight bearing added to a weak foot affected by arthritis always leads to pronation
- 3 Pain and peroneal muscle spasm with or without the additional factor of weight bearing may force the foot into valgus (pronation) Some relief from discomfort afforded by that position in acute cases induces the patient to maintain it
- 4 Spasm of the strong gastrocnemius muscle the effect of gravity and the weight of bedclothing pull the foot into a position of plantar flexion (equinus) (Fig 88)
- 5 The inflammatory periarthritic process at the metatarsophalangeal joints plus the effect of muscle spasm cause contracture of the extensors of the toes with depression of the heads of the metatarsals
- 6 Walking on such a forefoot with spastic muscles causes further plantar depression at the metatarsophalangeal joints

To prevent it

- 1 Relieve the foot of the strain of weight bearing as long as there is evidence of active arthritis The patient must be in bed (Fig 89)
- 2 The normal position of the foot—with correction of insidious valgus or equinus—may be maintained by a plaster boot or metal splint which may be removed for the application of physical therapy (Figs 90 91)

3 Protect the feet from weight of bedclothing by means of a cradle

Dr T Campbell Thompson has described and illustrated well some of the simple mechanical appliances which may be employed, when the pa-

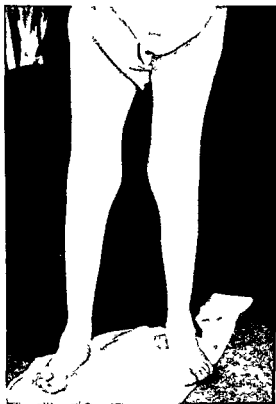


FIG 84 Mild degree of pronation of feet in a case of chronic atrophic arthritis. This patient also has moderate flexion deformities at the knees but has kept on walking because she was afraid of the joints getting stiff if she went to bed.

tient is in bed or ambulatory, to prevent deformity and relieve strain in the feet. Along with the illustration, which he kindly permitted us to reproduce, we quote from his description of the methods as follows:

A simple posterior wire splint is a very satisfactory foot support to prevent equinus (Fig 92, 1)

If there is a tendency toward claw toe deformity one can fit a special sandal with holes through the sole so that each affected toe may be strapped down against it (Fig 92, 2)

When the patient is allowed to stand, adhesive strapping for support and correction of pronation is usually very helpful (Fig 92, 3)



FIG. 85. Marked rigid valgus deformity of both feet in a case of long standing chronic atrophic arthritis in which all of the past treatment entirely disregarded consideration of these deformities at one time preventible or amenable to correction. Note also the marked thickening and valgus deformity of the left knee a condition undoubtedly aggravated by the static defect in the foot.



FIG. 86



FIG. 87

FIG. 86. Equinus (plantar flexion) deformity of both feet in a case of chronic atrophic arthritis caused by contracture of the gastrocnemius as a result of neglect of support of foot in normal position during the early stages of the disease.

FIG. 87. Plantar projection of the anterior (metatarsal) arch and cocked up toes in a case of chronic atrophic arthritis. This patient suffered agonizing metatarsalgia on walking because of weight bearing by the heads of the metatarsals and painful callosities over the forefoot.

Appropriate felt pads beneath the scaphoid bones or behind the metatarsal heads may be used to distribute the weight more evenly upon the sole of the foot (Fig 92 4)



FIG 88 Plantar flexion (equinus deformity) of the feet in the making as a result of pull of spastic gastrocnemius muscles aided by the effect of gravity

Sponge rubber or dug out heels are useful if the tuberosity of the os calcis is sensitive (Fig 92 5)

Elastic metatarsal straps with a metatarsal pad give welcome support and prevent spreading of the forefoot (Fig 92 6)

If the metatarsal heads are tender or the toes stiff and painful a large metatarsal bar often gives great relief (Fig 92 7)

For a weak arthritic foot with pain in the scaphoid region or along the inner side of the leg a well wedged Thomas heel with an outside iron band and an inside T strap is probably the best type of support (Fig 92 8 9)

- 4 When weight bearing is permitted after the activity of the process has subsided support the feet by proper shoes fitted properly The shank of the shoe should be strong enough to support the longitudinal arch Any sag of the longitudinal or anterior arches should be corrected by properly fitted plates or pads A straight last shoe—having a straight inner line from the longitudinal arch to beyond the great toe—is desired The shoe should be wide and long enough The heels of women's shoes should be broad and low

For correction of deformity if already present

- 1 Be sure that the patient wears good shoes, properly corrected, with plates or otherwise as described above in early flaccid stages

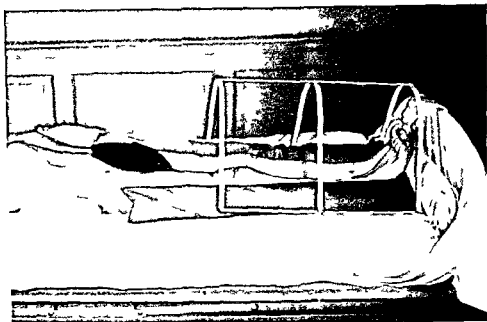


FIG 89 The patient in bed in a position aimed to prevent eversion and plantar flexion (equinus) at the foot. Note the sandbags at the sides of the lower legs preventing eversion of the legs and feet; the support at the base of the bed preventing plantar flexion; and a cradle over the feet to prevent the bedclothes from exerting their weight on the toes and feet.

- 2 Institute corrective exercises
- 3 Introduce gentle stretching, splinting and physiotherapy
- 4 Manipulate under anesthesia for correction of deformity caused by muscle shortening and capsular contractures, when the arthritis is quiescent. Follow manipulation by splinting, physiotherapy and corrective exercises.

Thompson outlines the operative correction of arthritic deformities as follows:

Hallux valgus and hallux rigidus are very common in arthritic feet. Removal of the exostoses from the metatarsal head and excision of the proximal one-half of the proximal phalanx usually corrects the deformity. The sesamoid bones and the weight-bearing area of the first metatarsal head should be avoided if possible, and an hour-glass constriction formed between the metatarsal head and the remainder of the phalanx to insure free mobility (Fig 93, 1).

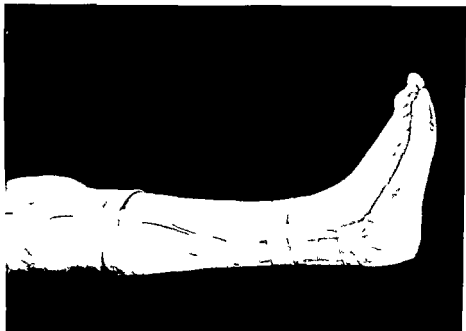


FIG. 90. A light plaster shell designed to prevent plantar flexion (equinus) at the ankle in a case of active atrophic arthritis. The splint is removed daily for the application of heat, massage, and exercises.

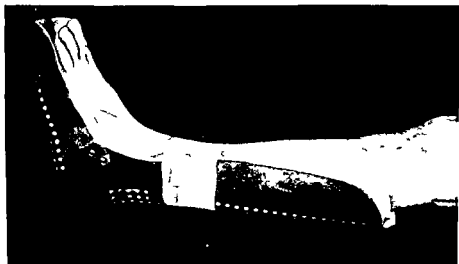


FIG. 91. A metal splint used to prevent equinus (plantar flexion) deformity of the foot.



FIG 92 Methods of preventing deformity and relieving strain 1, Osgood posterior splint 2, Claw toe sandal 3, Inversion strapping 4, Pads for metatarsal or scaphoid regions. 5, Sponge rubber for painful heels 6, Elastic metatarsal support 7, Metatarsal bar 8, Thomas heel 9 Outside iron and I strap (From Thompson, T C, *Medical Clinics of North America*, 21 1785 1937)

[Note In most cases complete excision of the metatarsal head is unnecessary Plastic operations on the periarthritic structures with or without osteotomy on the metatarsal frequently correct the deformity]

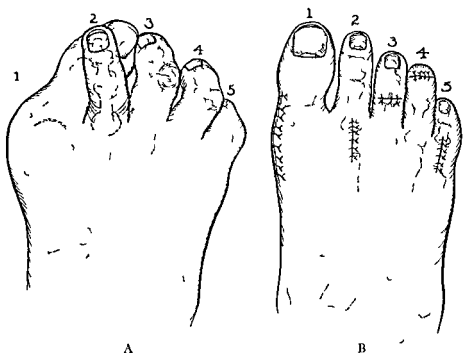


FIG 93 The operative correction of arthritic feet A 1 Hallux valgus and hallux rigidus (bunion) B 1 Hemiphalangectomy and exostosectomy

A Dorsal dislocation of second toe B 2 Excision of head of second metatarsal

A 3 Hammer toe with painful corn B 3 Excision of corn Fusion of interphalangeal joint (spiking operation)

A 4 Painful hammer toe with pressure on toenail B 4 Terminal Syme amputation removing toenail and distal phalanx

A 5 Claw toe projecting dorsally B 5 Excision of proximal phalanx (from T C Thompson *Medical Clinics of North America* 11 85 1937)

Any metatarsal head which becomes too prominent and bears an undue portion of the body weight should be cleanly excised through a dorsal incision allowing the weight to be distributed equilly to the other metatarsal heads (Fig 93 2)

Corns and calluses require continued protection against pressure or an operation correcting the bony abnormality which causes the pressure (Fig 93 3)

Hammer toe may be corrected by excision of the corn and arthrodesis of the deformed joint (Fig 93 4) However it is often advisable to

remove enough bone to allow free motion as arthrodesis in good position requires very prolonged and careful splinting and adds one more stiff joint to an already too rigid foot. In most operations upon the fore foot one should remove bone freely and soft tissue sparingly or not at all.

Severe claw toe especially if there is dorsal dislocation at the metatarsophalangeal joint can often be completely and easily corrected by excising the entire proximal phalanx which usually projects upward. This filleting operation permits the remaining bones to drop plantarward and the toe to assume a normal position (Fig. 93-5). The period of convalescence is very short and no immobilization is necessary.

Subcutaneous tenotomies of the extensor and flexor tendons of the toes and capsulotomies of the metatarsophalangeal joints will sometimes aid in the correction of deformed toes.

Amputation of one or even all the toes may be indicated if deformities are severe and the feet very painful. Equinus, cavus, varus and valgus deformities may require subastragalar, midtarsal or even ankle arthrodesis with the removal of appropriate bone wedges. The foot should be realigned beneath the leg with a large smooth weight bearing surface in contact with the ground. Painful motion should be eliminated from weight bearing joints by arthrodesis and from nonweight bearing joints by wide excision.

CHAPTER XVI

PHYSICAL THERAPY IN ARTHRITIS

Physical therapy in some form is recognized to be beneficial in all types and stages of arthritis. In the prevention and correction of deformities it is more than helpful, it is an integral part of local treatment. For centuries it constituted the backbone of therapy in the famous spas of Europe. To be sure many of the methods employed in the past were crude compared to those available today, but, if not curative, they at least afforded some measure of relief which was welcome, even when temporary.

We still have much to discover, of course, concerning the application of physical therapy in rheumatic diseases, but nevertheless we can secure excellent effects if we understand and apply precisely the facts at hand. The amount of heat, the type of massage desired, the place where it is most needed, the appropriate or inappropriate time for manipulation—these are only a few of the recommendations the physician should be able to make to the physiotherapist, who, in turn, should be competent to execute them. Incidentally, proper training and experience on the part of the physiotherapist is of far greater importance to the success of treatment than glittering and expensive equipment. Obviously, under proper circumstances, there are certain benefits to be derived from the employment of the more elaborate methods, such as diathermy, faradism, actinotherapy, and deep x-ray therapy, but fortunately for the many patients for whom these means are entirely out of reach, simple, inexpensive physiotherapeutic methods seem hardly less effective when properly employed.

There is an additional advantage in using simple, inexpensive physiotherapeutic methods; they can be easily applied in the patient's home (where the treatment must be carried out) to supplement that available from the professional physiotherapist.

The physiotherapeutic means chiefly employed in the treatment of arthritis are heat, massage, and exercise. Since this book is addressed to the physician in general practice, only the simplest and most easily available methods for procuring these therapeutic aids will be discussed here; the more specialized techniques that may be employed when available will be mentioned briefly. For further details the reader is referred to a recently

published monograph by Krusen, dealing exclusively with physical therapy in arthritis

HEAT

Heat applied locally to the affected joints, is helpful in practically all types of arthritis and in fibrositis. Its most evident effect is to increase the flow of fresh blood in the affected tissues by means of local vasodilatation. It is generally better tolerated by joints affected with hypertrophic arthritis than by those with the atrophic type, in which heat may be overused during the active more acute stages of the disease. However, in the more chronic stages of atrophic arthritis, when heat is more necessary, it is well tolerated. The initial period of exposure to heat should be not longer than fifteen minutes in the average case. This is particularly important in the more acute phases of atrophic arthritis. The effect is then observed, and, if conditions warrant the period of exposure may be increased gradually to twenty or thirty minutes. As with any potent medicinal agent, the dosage prescribed may require modification from time to time. When many joints are affected and require heat locally, the treatments may be spread out over several periods during the day, otherwise the period of exposure for each joint would have to be shortened. In some patients, following too long application of heat to many joints, the treatment induces a systemic debility more serious than the local condition. Old patients with hypertrophic arthritis, in whom many joints may require baking, may be less tolerant of the heat than of their condition. These facts must be considered and the patient observed, if harm is not to be inflicted.

The ordinary tungsten filament lamps emitting luminous and short infra red rays are more effective in increasing the temperature of the subcutaneous structures than the usual infra red generators or carbon filament lamps, which predominate in rays that penetrate less deeply, and are, furthermore, more expensive and less easily procured.

Krusen has described a simple, inexpensive lamp which can be assembled for several dollars. It consists of a large heat bulb, a polished reflector, and a clasp for attachment to a chair or a bed.

The author ordinarily prefers to use a simple home made electric light baker (Fig 94) with four to eight bulbs. These are inexpensively made by a tinsmith who follows the specifications described in a leaflet issued by the Council on Physical Therapy of the American Medical Association. The specifications for this baker are as follows:

17 inches long

14 inches wide

14½ inches high over all. If the baker is to be used for the body, as well as for the legs or arms, supports should be two or three inches longer.

Altitude of arc—5 inches

Frame— $1/16 \times 5/8$ inch strap iron

Reflector—highly polished tin sheeting

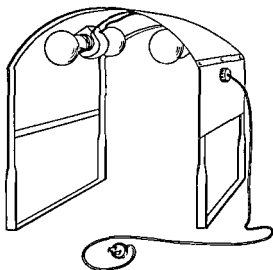


FIG. 94 Home made baker for therapeutic use designed for applying heat to the legs and arms or the body

Two to 4 double receptacles General Electric Company catalogue 66722
250 volts, 650 watts

Four to 8—60 watt mazda lamps

The tin is riveted to the strip iron. Receptacles connected in multiple with heavy lamp cord 6 feet long. Hubble plug at the end of cord.

When some form of heat lamp is not available, the local application of hot paraffin to the affected areas may serve as an excellent substitute. Krusen, in his book on physical therapy in arthritis, describes the procedure as follows:

The patient is instructed to purchase at the grocery store 6 to 8 pounds of paraffin (such as is used to seal jelly and fruit jars). This is placed in the inner pan of a double boiler (such as is used to cook cereals), and the outer portion of the double boiler is filled with water. The whole is placed on the kitchen stove and heated until the paraffin has melted. It is then cooled until a thin scum of cooling paraffin is beginning to form on the surface. At this time the paraffin will be at its low melting point, which is the high heat tolerance point of the human skin. A clean paint brush or wooden paddle covered with gauze is then used to paint about eight layers of hot paraffin on the affected joints or other areas of the body surface. This thick layer of paraffin is left in place for one half hour and then may be lifted off readily in a single sheet and later remelted for subsequent use. An excellent hyperemia will be produced by this

method The paraffin should not be applied over very hairy skin without previous shaving Rarely a patient's skin may be sensitive to paraffin In such cases mild dermatitis may develop For the most part we have found this to be an excellent method for accurate application of heat to arthritic joints when electricity is not available

The following procedure is employed to apply hot paraffin to the hands or feet The paraffin is melted in the manner just described It is then poured into a pan large enough to accommodate the hands or feet which are then immersed in the melted paraffin and removed the paraffin solidifying when exposed to the air This procedure is repeated six times in quick succession before the paraffin has cooled too much until a moderately thick glove-like coating is produced The extremities are then wrapped in a towel for twenty five minutes following which the paraffin is peeled off and saved for further use

Hot compresses made of light turkish towels saturated with a solution of hot magnesium sulphate (epsom salt) with the excess solution wrung out may be applied when none of the other methods of applying heat is available Hot epsom salt compresses may be particularly useful over acutely inflamed swollen joints

The hot water bag or any other type of application of heat may give some temporary relief from soreness and stiffness but these procedures do not afford deep penetration of the heat and are, therefore not as useful as radiant heat for stimulating vasodilatation Immersion of the hands in hot water is another means of securing this effect

Contrast baths that is immersion of the hands and feet in alternately hot and cold water may be useful in improving peripheral vasomotor tone Some persons respond poorly to contrast baths noting more stiffness after such a procedure than before These are generally patients with extremely poor peripheral capillary circulation which is handicapped still further by the cold phase of the contrast bath They generally respond favorably to hot baths or to applications of hot paraffin For the contrast bath the patient employs two containers one of which is filled with hot water (110°F), the other with cold (60°F) The extremities are immersed for one minute alternately in the hot and cold water for a total of fifteen minutes the procedure beginning and terminating in the hot water

Systemic Exposure to Heat

Systemic exposure to heat by means of hot baths hot packs or other procedures may relieve nervous and mental tension stimulate general metabolism improve the general circulation and if the body temperature is elevated produce leucocytosis of some degree

It must be remembered however that the patient must be chosen with

care, for one who is debilitated may be completely prostrated by over exposure. Always, the patient should be exposed for only a brief period at the outset, and should be observed carefully for the effect both during the period of exposure and afterward. If such hydrotherapeutic measures produce undue debility they are obviously not serving a useful purpose and should be discontinued.

The effect of systemic exposure to heat, as in a simple hot bath, may be enhanced by contrasting brief exposure to a cold shower immediately afterward.

There are still other advantages that may be derived from simple hydrotherapeutic procedures as for example underwater exercise of sensitive joints, which could not tolerate motion otherwise.

We have merely outlined the various simple methods for the application of heat to indicate how readily these procedures are adapted for use in any hospital and in most homes. For detailed descriptions of the methods and problems met in the actual application of these measures the reader is referred to Krusen's manual on "Physical Therapy in Arthritis." Written for the general physician, the instructions are brief, specific, and the procedures so simple as to be applicable in the home, as well as in the physician's office or hospital.

It is generally accepted that physiotherapeutic measures in arthritis, limited to one or two treatments a week in the physician's office or hospital, are grossly inadequate. In most cases more frequent use of professional physiotherapy is impossible because of the inconvenience or expense involved. Provision must therefore be made for training the patient and some members of the family in simple, harmless, but effective physiotherapeutic methods which can be carried out daily in the patient's home. Actual experience has proved that simple physiotherapy, carried out at home, after some preliminary training and with constant supervision and guidance by the physician or professional physiotherapist, can bring gains which would be impossible otherwise. It is significant that Krusen, heading an institutional department of physical therapy in which practically every useful physiotherapeutic appliance and skill is available, concludes a discussion on physical therapy in arthritis with the advice that "with a little ingenuity the family physician can supply nearly all the necessary physical therapy in the patient's own home."

Diathermy

Diathermy is another means of applying local heat to joints. It is agreed that it has no specific biological effect other than supplying heat, although it is true that better heating of deeper tissues is effected. For this reason it may be considered superior to radiant heat when used in more indolent

stages of chronic atrophic arthritis in hypertrophic arthritis, or fibrositis. For the same reason, however, it is less useful in the more acute stages of atrophic arthritis in which radiant heat affords some relief from congestion and pain by causing more superficial vasodilatation, with reflex vasoconstriction in the deeper structures of that vicinity. Requiring elaborate and expensive apparatus, it is not so readily available and is more expensive than heat obtained in other ways. The important fact is that it is not indispensable; adequate physical therapy in arthritis is not dependent on possession or even the best use of a diathermy machine.

Short wave diathermy, the newer method for the application of deep local heating, may have some advantages over the older methods, but is again essentially merely another method. Krusen recently reviewed the present status of short wave diathermy and indicated its logical place in the treatment of arthritis and other conditions.

Inductothermy

Inductothermy is still another method for supplying deep local heat. It has, if anything, certain advantages over diathermy, in that a maximum of heat is created in vascular tissues where conduction is easiest, and a minimum in bone and adipose tissue where it is most difficult. This is the reverse of the effect of diathermy, in which heat is generated by the resistance of the tissues. Treatment is administered by means of a flexible cable or a disc. Since surface electrodes, such as are required in diathermy, are not used, the risk of superficial burns is largely obviated. The physiologic effect of inductothermy is otherwise quite similar to that of diathermy.

HISTAMINE AND ACETYLCHOLINE IONTOPHORESIS

Although the introduction of such vasodilators as histamine and acetylcholine by means of the galvanic current—by iontophoresis—has been advocated and may be useful, one may doubt whether the average patient with arthritis is justified in trading the known benefits of the simple, daily rite with an ordinary baker, for the more occasional, expensive, and elaborate ritual of iontophoresis.

MASSAGE

The primary effect of local massage in arthritis is to increase local circulation by opening vascular channels. It is practically always administered after the application of heat to the area, the massage actually carrying a step further the circulatory action started by the heat. Through the mechanical influence on the circulation of the tissues massaged, improved local

metabolism, possibly some resorption of exudate, reduction of muscular pain and soreness, and preservation or restoration of muscle tone results. To accomplish these things the massage must be administered properly, for otherwise it may injure rather than aid, retard rather than advance progress. Nowhere is gentleness and thoughtful consideration of the tissues treated so imperative as in the administration of massage or exercise to the patient with arthritis. It is so easy to overdo the massage and thus add the effect of trauma to that of inflammation.

The tissues massaged are, of course, chiefly those around the joint and not the joint itself. Unless this is made clear to the patient he is most apt to do just the reverse and the more swollen the joint, the more diligently he may massage it. Although *light superficial stroking* directly over the joint may be of benefit in chronic arthritis it should never be attempted when the joint is acutely inflamed. Even the muscles above and below the joint should be given very light massage, at first for but a few minutes. More vigorous massage should not be instituted until the tolerance for it becomes evident, however, it is tolerated and distinctly beneficial in the less active stages of atrophic arthritis, in most cases of hypertrophic arthritis, and particularly in the treatment of fibrositis (muscular rheumatism). Deep massage, with an attempt to break up palpable, painful fibrositic nodules has been especially recommended by some clinicians.

General body massage may somewhat ameliorate the effect of inactivity in a patient confined to bed and may also contribute to the improvement of the physiologic status in other ways. However, it should never be employed on acutely ill patients, and should preferably be instituted after a period of treatment with local massage, during which the tolerance of the patient has been appraised.

The effect of massage is constantly cumulative. No significant benefit will result from a more or less casual treatment administered once or twice a week. Daily massage (preferably twice daily for short periods) must be provided. Again, home treatment becomes essential for most patients. Some interested member of the family should be instructed in the use of massage, of the type indicated in the given instance. The treatment thus provided may not be as expert as professional massage, but it is better than none, and far better than the "rubbing" the patient is liable to adopt for himself, without such guidance.

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Exercise heightens enormously all of the physiologic effects of massage, and provides additional benefits not available through massage alone. In a previous chapter dealing with the prevention of deformities we stressed

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EXERCISE

Exercise heightens enormously all of the physiologic effects of massage, and provides additional benefits not available through massage alone. In a previous chapter dealing with the prevention of deformities we stressed

the importance of motion in joints to prevent ankylosis and to retain the integrity of muscular power. In that connection we referred frequently to the employment of exercise as early as possible and to the extent that the condition of the joints permits. When motion is very painful and limited by activity of the inflammatory reaction in the joints we fall back upon massage as the temporary mainstay although even then passive exercise should be attempted and is beneficial. As the inflammatory process wanes the effect of massage is augmented by active exercise which is constantly though gradually increased in range and frequency as conditions permit. The increased circulatory flow induced by massage is negligible in proportion to that effected by active motion; the same applies to the matter of local tissue metabolism and through it to heightening of the systemic metabolic fires. The relief of muscle soreness and stiffness—the limbering up process—of which the patient always tries to avail himself in the morning after nocturnal inactivity is probably largely the result of accelerating such circulatory and metabolic processes by exercise. But most significant for the maintenance of the integrity of muscles and for their regeneration if atrophy has occurred is the effect of active muscular contraction.

Passive motion occupies a place midway between massage and active exercise so far as its physiologic effect is concerned; active exercise against resistance properly applied accentuates the effect.

As with other physiotherapeutic measures the proper adjustment of the type, grade and extent of active exercise to the needs and capacity of the patient is important. The limbering up process which brings so much relief is generally interpreted by the patient literally as the sign to keep going in order to maintain the relief afforded in the afternoon hours. But to his disappointment he learns soon that for the few grateful hours of relief he may pay with hours of fatigue and pain in the evening and the next morning. Thus also therapeutic exercise when overdone may be followed by relief immediately afterward and by increased soreness, stiffness or pain the next day. One must be as alert to the dangers of an overdose of exercise as to those of an overdose with potent medicinals. Instead of accelerating muscular development the fatigue of overexercise is likely to deter progress both locally and systemically.

Active exercises may have to begin with movement of single muscles to be extended to muscle groups and later to the body as a whole.

Earliest active motion is encouraged in atrophic arthritis where the tendency to muscle atrophy and ankylosis is so prominent a factor in the disease. In hypertrophic (osteo-) arthritis in which ankylosis is rarely a factor to contend with early active exercise is not so urgent. The patient may indeed benefit from deferring active motion for a while in order to permit

resolution of the effect of former trauma. The development of muscle atrophy in the patient with hypertrophic arthritis is furthermore a slow process seldom of significant degree and seemingly dependent entirely on the factor of disuse. In atrophic arthritis there must be some sort of disturbance in the trophic regulation of muscle tone and integrity and this may assert itself with lightning rapidity and with devastating effects even before the factor of muscular disuse inflicts much of its own damage. Possibly for the same reason we find that muscular atrophy of atrophic arthritis responds more slowly to the same therapeutic measures which are effective so much more rapidly in other types of atrophy.

The possibilities of underwater exercise have already been mentioned. These may be attempted with some facility by patients during the early stages of convalescence when active motion would otherwise be much more difficult. The combination of hydrotherapy and active exercises is most applicable in the physiotherapeutic after treatment of joint deformities which have been corrected by manipulation or by surgical means.

OCCUPATIONAL THERAPY

Occupational therapy is an ideal extension of the principle of active exercise and is an interesting and pleasant way of administering the latter.

LOW FREQUENCY CURRENTS

The interrupted galvanic or faradic current may be employed advantageously to stimulate passive muscular contractions before the patient has the power for active exercises. It is also efficacious temporarily when passive muscular contractions are desired while joint motion is impeded by pain and it is useful simply to demonstrate to the patient what muscular contractions he should attempt himself. The effect of passive electrical stimulation of muscular contractions is hardly to be compared with that of active motion which should always be encouraged. If nothing more is possible active muscular contractions without joint motion should be attempted.

POSTURAL REHABILITATION

Abnormalities of body posture frequently of severe grades are among the most frequent stigmata of the patient's makeup. Such postural disturbances are evidenced most glaringly in the thin long-chested asthenic

viscerotonic types of individuals with atrophic arthritis. As Osgood says of these persons: "The set up is inefficient. Their muscles are poor, their thoracic cages are narrowed, their diaphragmatic excursion is small, their abdominal viscera are sagged, the weight bearing lines of their joints are not true, muscle tonus is hard to maintain because the center of gravity is disturbed. They are fatigued. Their body mechanics is wretched."

Much of this is probably the result of an inherent weakness in body structure intensified by fatigue, poor body carriage, infection and other extraneous influences. In turn, the poor posture aggravates the tendency to fatigue and the susceptibility to infection. A vicious circle is provided which permits the onset of the arthritic disease and militates against recovery.

The beneficial effects of correct body posture have been accorded too limited recognition. Obviously, postural correction plays a less striking part in the treatment of atrophic arthritis than it can play in its prevention. In the one already afflicted, postural rehabilitation may, however, be the determinant of complete recovery and freedom from recurrence.

The correction of faulty posture must begin with correction of joint deformities, if present. The elimination of fatigue by adequate rest is of equal importance and correct postural attitudes should be assumed during the period of rest. The position of dorsal hyperextension is most important. The patient lies on his back with a pillow under the dorsal spine to decrease the dorsal kyphosis. By thus increasing the capacity of the thorax, he induces better circulatory and pulmonary function. Such a position is assumed for short periods of fifteen to twenty minutes at intervals during the day. A sagging position in bed should be avoided by means of a rigid (though comfortable) mattress and spring. This may be accomplished by placing a board between them.

Simple postural exercises are begun in bed, the patient graduating by degrees to other postural exercises in the erect position. Those which may be required are detailed specifically in Thomas and Goldthwait's book on *Body Mechanics and Health* and in Goldthwait, Brown, Swain and Kuhn's book on *Body Mechanics in the Study and Treatment of Disease*. Realizing the importance of this aspect of their practice, most physiotherapists are eager for the opportunity of spreading the gospel. Indeed, the professional physical therapist is ready to lend a very helping hand to the practitioner who is without the time or inclination for instructing his patients and for observing their accomplishments.

In hypertrophic arthritis, too, affecting the more sthenic type of individuals, postural abnormalities are not infrequent. In these cases, trauma of obesity frequently contributes to local postural disturbances and strain in

weight bearing areas, with resulting repercussions on the body structure as a whole. Again, postural abnormalities resulting from poor body carriage may predispose the cervical and dorsal spine to osteoarthritis and may aggravate the symptoms which result.

Postural re education in older subjects, those susceptible to hypertrophic arthritis, has various limitations. The age of the individuals affected is a deterrent to such a plan, the very nature of the pathologic process produces intolerance of exercise and need for rest and immobilization. In many of these patients, particularly the older ones, palliative and supportive measures which improve the body mechanics suffice to ameliorate the strain of bad posture. Thus, corrective devices for the feet, reinforced corsets and jackets, back braces, cervical collars, and other such appliances, may be required to correct certain of the postural defects which are contributing to the discomfort induced by hypertrophic arthritis.

HELIO THERAPY

Exposure to sunlight, natural or artificial, may be employed with benefit, if used intelligently. It must be made clear to patients that sunshine, whether obtained naturally or from artificial sources, is not a cure all. It cannot be exclusively depended upon to eradicate the arthritis unless it is used in conjunction with all other essential therapeutic measures. For local heating effects, infra red radiation cannot be supplanted by ultraviolet. When the latter is employed it should be chiefly for its effect in stimulating general metabolism and perhaps synthesis of vitamin D. Over stimulation of metabolism must be avoided. In acutely ill and debilitated patients over exposure may be distinctly injurious.

There are practical obstacles to the employment of heliotherapy. To the largest proportion of patients with arthritis, natural sunshine is not always available and effective artificial heliotherapy apparatus is expensive. No one need despair because heliotherapy is not available, it certainly is not one of the essential agents of treatment in arthritis.

THERAPY WITH RADIO ACTIVE SUBSTANCES

The use of salts of radium and of other radio active substances in arthritis is mentioned here merely to emphasize the point that their effect is not specific, their value unproved, and the possible dangers attendant upon their use serious enough to limit their usefulness, for the present, to the cautious experimenter. Radium waters, exploited from time to time for the treatment of arthritis, are dangerous, if not ineffective.

X RAY THERAPY

Roentgenotherapy used in special cases for many years especially in European clinics has in recent times received more extensive trial in various types of arthritis. Its specific action, its place in the therapy of arthritis and the basis for selection of cases are still far from clear. There appear, however, certain apparently reliable studies on the subject which indicate that x ray therapy may be justifiably accorded a place among the more special (if more restricted) methods of treatment in certain types of rheumatic conditions. Obviously, this form of therapy requires very specialized apparatus and expert technical application. Without the latter it is a distinctly hazardous procedure.

One of the best reviews on the subject of roentgenotherapy in arthritis is that by Gunnar Kahlmeter of Stockholm. He submits also a rather favorable impression of x ray therapy gained from his own and his colleagues' experience in the treatment of many thousands of cases over a period of twelve years. For the benefit of those who may be interested I shall quote some of the salient features of Kahlmeter's report. For the roentgenologist who may be called upon to apply such therapy I shall quote some of the technical details of dosage recommended by Kahlmeter. The original publication when available might be profitably consulted as might also those reports which give a less favorable impression on the efficacy of roentgenotherapy in arthritis.

Kahlmeter describes his present technique and therapeutic plan for various types of arthritis as follows:

The tube output is 173 kw (kilowatt) at a current of 6 ma (milliamperes) and the filter used is equivalent to 0.5 Cu + 1 Al. The focal distance is 50 cm except for fields the size of 15 x 20 cm where we use 40 cm. The field generally used measures 10 x 15 cm although 8 x 10 cm is also employed. With the above technique 1 SLD in 1 field of 10 x 15 cm is equal to 1000 r.

In the table showing our system of dosage I have put down not only the dosage used in different forms of chronic arthritis but also that used in lumbago and neuralgia, acute bursitis, more chronic forms of tendinitis in humer scapular periarthritis and in gonorrhoical arthritis.

The size of the irradiated field varies according to the size of the joint. That mostly employed measures 10 x 15 cm but 8 x 10 cm and 15 x 20 cm are also used, the latter particularly in the case of root fields in neuralgia and for osteoarthritis of the hip and spine. In chronic lumbago on the other hand localized to the tendinous insertions of the erector spinae musculature or the sacroiliac articulations smaller fields are used. When the entire hand (wrist, metacarpophalangeal and interphalangeal joints) is being irradiated large fields are used.

TABLE OF DOSAGE

	SINGLE DOSE	DOSES IN ONE SERIES	INTERVAL BETWEEN APPLICATIONS IN ONE SERIES
	r	Number	Days
Lumbago	150 (1/6-1/7 SED)	2-3	2-3
Neuralgia sciatica, and brachial neuritis	150-200 (over root field)	2-3	2-3
Acute bursitis and peritendinitis calcarea (subdeltoid, subacromial etc)	200	1-2	2-3
Tendinitis (trochanteric, pes anserinus, calcanei, tennis elbow etc)	150	2-3	2-3
Humer scapular periarthrits (omarthrits)	150	2-3	2-3
Acute gonorrhoeal arthritis	75-100	3-4	2-3
Rheumatoid arthritis	100-150	2-3	2-3
Osteo arthritis of the hip-joint	150-200	2-3	2-3
or sometimes in old cases	200	5-6	7
Osteo-arthritis of the spine (spondylitis deformans)	200	5-6	7

15 x 20 cm, on the dorsal aspect only. Wrists, elbows, shoulder joints and knee joints are treated over two different fields, hip joints over three. We have given up irradiating three fields in cases of shoulder joints and knee joints, since "overlapping" which may involve unnecessary risk of damage, may then be difficult to avoid.

To avoid skin reactions from the x rays Kahlmeter observes at least one week's rest, before and after x ray irradiation, from all forms of treatment producing hyperemia of the skin (hot air, light diathermy, short wave treatment, ointment dressings, plasters and so on).

As to the results of treatment reported by Kahlmeter, we shall quote him again as follows:

I think that undoubtedly the best results of x ray treatment are obtained in gonorrhoeal arthritis, articular gout in the subacute stage, acute septic (infective) arthritis, acute bursitis and tendinitis. Furthermore, the results are very

good on the whole in all forms and stages of tendinitis and periarthritis in localized myalgia and neuralgia and on the whole in all cases with periarthritic symptoms. This last mentioned factor is of importance when it comes to choosing the most effective radiation technique in the treatment of different forms of chronic arthritis where it is easy to understand the results are not and can not be as satisfactory as in the first mentioned groups of disease.

With regard to the effect obtained in gonococcal arthritis it must be emphasized that Kahlmeter usually resorts to fever therapy along with x ray therapy and fever therapy alone is admittedly extraordinarily effective in the largest proportion of cases of early gonococcal arthritis.

An excellent result of x ray irradiation is also obtained in the acute forms of bursitis. In some cases however particularly in those of less acute onset or even those running a chronic course the clinical picture merges imperceptibly into those conditions I have called peritendinitis tendinitis and even in some cases periarthritis. In all these different localisations and forms of peritendinitis and tendinitis the effect of x ray therapy is very good even if not so striking and above all so rapid as in acute bursitis. I should think the figure given in 1932 for good results free or nearly free from symptoms (90 per cent) is well maintained.

In my opinion the favorable result of x ray we in many cases observe in spondylitis deformans is mainly to be ascribed to their beneficial effect upon the irritation and tenderness here of the deep attachments and aponeuroses of the entire lumbar musculature.

Likewise I am convinced that most pains in osteo-arthritis of the hip-joint are caused not immediately by changes in the joint itself but by hyperfunction of the muscles of the hip thigh and back which is the result of restricted mobility in the hip-joint. It seems to me scarcely possible to explain the therapeutic effect attained by x rays in this disease in any other way than by some direct influence of the rays on the painfulness localized to all muscular attachments round the hip-joint not least to those of the abductors and adductors of the thigh. Exactly the same applies to osteo-arthritis of the knee joint the chief effect of irradiation can probably be referred to the tendinitis so often present in the attachments of adductors and the tendon of the quadriceps and particularly in the medial tendons (pes anserinus). In climacteric arthritis of the knees where the symptoms mainly consist of such tendinitis and periarthritic tenderness and swelling the effect of x ray therapy is extremely satisfactory.

Concerning the results obtained in atrophic arthritis Kahlmeter says

The task of estimating the value of x ray therapy in this disease is very difficult indeed. Numerical figures here indicating the results are of exceedingly slight value because naturally the effect of treatment varies so enormously with different stages of the disease and with different clinical types. Obviously no therapeutic action can be expected on destructive processes in cartilages on

ankyloses or anatomically fixed contractures. Nor is there any great effect on intra articular exudates, except in very acute and recent cases. A better result is obtained on inflammatory processes in the joint capsules, provided they are not yet organized, but the best effect by a long way is obtained on periarticular connective tissue, in tendon sheaths and attachments of tendons and muscles. The effect here approaches that earlier mentioned concerning sporadic bursitis and peritendinitis of a different etiology. In summary it may be said of the x ray therapy in rheumatoid arthritis that its effect is pronounced in the same degree as the "joint" symptoms are of recent nature, clearly inflammatory, and periarticular, and the restricted mobility is due to painfulness of the periarticular soft parts, and not to cicatricial shrinking, effusion or destruction of cartilage.

It will be noted from a close examination of Kahlmeter's report that the best results of roentgenotherapy occur in the most acute stages of bursitis, in various acute types of specific infectious arthritis, and in the more acute phases of atrophic arthritis when the symptoms are attributable to purely periarticular inflammatory changes. These are the forms and stages of rheumatic processes which respond most favorably to every other type of simple, accepted therapy, processes which, in fact, may clear spontaneously if nature be given a bit of support. In the more chronic forms of fibrositis ("tendinitis"), where other forms of treatment may be disappointing, an earlier trial of roentgenotherapy would be justified. But, in the vast majority of the more acute rheumatic conditions, the physician may confidently resort to simpler, more firmly established forms of physical therapy before resorting to x ray treatment.

Even those authors who are enthusiastic about roentgenotherapy in arthritis stress the point that "the therapeutic effect is entirely confined to the subjective symptoms, aiding return to normal function and ameliorating inflammatory symptoms if present." In certain cases the relief from pain may be merely the result of the analgesic effect of x rays. The possible destructive influence of x rays on cells of the inflammatory exudate may contribute to the amelioration of the signs of inflammation.

The author's experience with roentgenotherapy is too limited to justify even tentative conclusions, but despite those quoted, it is my opinion that, until more definite proof of its place and its value appears, roentgenotherapy should be used conservatively in the treatment of arthritis. We have discussed at some length the place of roentgenotherapy in the therapeutic scheme of arthritis because this subject has received, perhaps, too little notice in the American literature. Roentgenotherapy as a means of alleviating arthritic conditions deserves further study by those equipped with the clinical material and the technical facilities to carry out a research program.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527]

PART III

HYPERTROPHIC ARTHRITIS

HYPERTROPHIC ARTHRITIS SOME GENERAL CONSIDERATIONS

PATHOLOGY OF HYPERTROPHIC ARTHRITIS

CLINICAL MANIFESTATIONS OF HYPERTROPHIC ARTHRITIS

TREATMENT OF HYPERTROPHIC ARTHRITIS

CHAPTER XVII

HYPERTROPHIC ARTHRITIS

SYNONYMS *Osteo arthritis, degenerative arthritis, senescent arthritis, climacteric or menopausal arthritis (in women), osteo-arthritis, arthritis deformans*

Hypertrophic arthritis is to the joints what arteriosclerosis is to the vascular system, essentially a manifestation of the wear and tear of living, each is a price nearly all of us must pay for living long enough, for with age, some degree of these changes is inevitable

NOMENCLATURE

"Hypertrophic arthritis" is the term accepted by the American Rheumatism Association. Others have applied the designation "senescent arthritis" to some of those cases that fall within this large group, because senescence is an important factor in this disease. The condition may be brought on by degenerative changes in the joint cartilage (as Bennett and Bauer have recently shown), hence the name "degenerative arthritis." The British, to stress the early bone involvement in contrast to the primary periarthritic changes in atrophic arthritis, have preferred the term "osteoarthritis." The designation "osteoarthritis" has been employed to denote the absence of an inflammatory basis for this form of joint disease. "Climacteric" or "menopausal" arthritis has been employed by those authors who believe that the menopause exerts a precipitating influence in some cases occurring in women at the climacteric. "Arthritis deformans" is altogether too loose and meaningless a term for use.

ETIOLOGIC FACTORS

Long exerted trauma of mild degree, or more severe and abruptly inflicted trauma, is the factor most prominently related to the etiology of hypertrophic arthritis. Apparently even physiologic trauma may produce Heberden's nodes at the distal joints of the fingers or hypertrophic lipping in the knees of obese patients, but these changes are rarely seen in patients

who do not also show evidence of general physiologic aging or premature physiologic senescence

Because some individuals may develop hypertrophic arthritis at a relatively early age and others escape almost entirely significant degrees even in old age it is evident that senescence is not the sole factor concerned. This suggests the existence of an inherent relative immunity in some individuals and constitutional predisposition in certain others these latter possibly being born with tissues poorly equipped to withstand the burden of the ordinary wear and tear of living

Clinically the inherent tendency to this form of arthritis is suggested by the frequent incidence of the same condition in at least a number of the patient's familial antecedents. The patient himself may recognize and point out the genetic relationship between his disease and that of his ancestors when he volunteers as he sometimes does a comparison of his present condition with that of the parent who was similarly affected. Such an hereditary relationship is most often observed with reference to the occurrence of Heberden's nodes

This predisposition to primary degenerative changes in joints must be dependent upon a poor quality of the cartilaginous and bony structures and it is probably influenced by a poor vascular supply which becomes progressively more restricted with advancing age. Goldhaft, Wright and Pemberton have demonstrated that experimental ligation of a large part of the vascular bed around the patella in dogs brings about changes identical with those in hypertrophic arthritis of man. Studies of Heberden's nodes removed from affected fingers reveal some narrowing of the blood vessels to account for a decrease in the amount of available circulation. This of course may be only a part of the general narrowing of the circulatory bed which accompanies advancing age. If as Osler has said a man is only as old as his arteries then this form of arthritis so closely associated with physiologic age may be merely a concomitant of aging arteries in the joints.

It is not in the least surprising that trauma may add an intolerable burden to joints inherently inferior in structure. Even physiologic activity may be too much for them. One may well wonder whether the ubiquity of Heberden's nodes is not to an extent influenced by physiologic use of the hands. Such a view is largely substantiated by the occurrence of Heberden's nodes in the fingers of the nonparalyzed active hand of a patient with poliomyelitic paralysis of the other hand.

Bennett and Bauer demonstrated that degenerative and hypertrophic changes identical with those observed in man can be produced experimentally in rabbits by simple patellar displacement. Following such an operation which introduces a factor of mechanical stress there first appear degenerative changes in the hyaline cartilage then hypertrophic lipping

(spurs) at the margin of the joint surface of the bone. Such proliferation of bone occurred at the point of reflection of synovial membrane to the perichondrium of the articular cartilage, where these tissues are always exposed to physiologic stress. Excessive strain and the abnormal anatomic configuration within the joint accentuate the tendency to proliferation of this vascular fibrous tissue. It later becomes transformed into fibrocartilage or hyaline cartilage, and finally into bone. Marked hypertrophy of the synovial membrane also was noted and in one animal loose bodies were encountered. These changes were limited to the knees in which patellar displacement has been induced; they were absent in those which served as controls. It is clear from these experiments that localized trauma, without concomitant systemic disease, can produce the changes of hypertrophic arthritis.

Trauma from Faulty Postural Alignment

The chief contributory form of trauma is that resulting from strain of poor postural alignment. The most common of such abnormalities are pronated feet, flattened longitudinal arches with foot strain, depression of the anterior (transverse) arches of the feet, and knock knees. Any of these static defects or a combination of them may accentuate a tendency to hypertrophic arthritis and induce thickening of the synovia, particularly a villous synovitis of the mesial aspects of the knees. Postural errors may occur in thin persons whose musculature is flabby, but are particularly likely to develop in those who are obese.

Obesity

Microtrauma of obesity is another factor often bringing out the latent susceptibility to hypertrophic arthritis. In such cases the condition may be confined to weight bearing joints, particularly the joints in and about the lower spine, the knees, and feet. It is not difficult to imagine the strain these joints suffer from the constant weight of a load for which they are not prepared. What probably occurs first is a distortion in the architectural alignment of the bony structures, resulting in abnormal distribution of weight on the joints. Constant repetition of the pounding on the abnormally situated joint structures results in infinitesimal injury, which accumulating over periods of years produces the final result—hypertrophic arthritis. The changes in the joints actually reveal such a progression. Since the damage is of relatively mild degree, the proliferative process in the joints proceeds at a progressive, if slow, pace. The result is an overgrowth of the joint margin with irregular bony projections and thickening of the synovial lining. This is most evident in the knees, where an irregular, marginal proliferation is noted at the joint surfaces of the bones. In the spine,

similar sharp projections can be seen extending from the edges of the vertebrae

The drag of a pendulous, obese abdomen exerts strain not only on the ligaments and joints of the lumbar spine, but also on the dorsal spine, because of the compensatory dorsal kyphosis which results. In turn, postural strain may result at the cervical spine when the head is thrown forward to compensate for the exaggerated lumbar lordosis and increased dorsal kyphosis. Other effects of the excessive weight are exerted on joints which bear the greatest load—the ankles and the joints of the feet. The ankles become thickened, the feet pronated, and the arches flattened and painful. Indeed, the association of low backache with pain in the knees, ankles, and flattened arches will usually be found to be due to the strain and trauma of obesity and hypertrophic arthritis.

Occupational Trauma

The effect of occupational trauma is manifest by the tendency to Heberden's nodes among stenographers, gardeners, and those who knit and crochet excessively. The cervical spine may also be subjected to postural strain resulting from certain occupations, thus, it may be a cause of much trouble among bookkeepers, teachers, taxicab drivers, and card players.

Foreign Bodies

Degenerative and hypertrophic joint changes and synovial hyperplasia may develop when a foreign body enters or becomes lodged near a joint and remains there for a length of time. If the foreign material is not promptly removed, the joint may become totally disorganized and hopelessly damaged. Degenerative changes in the cartilage, secondary hypertrophic changes both at the joint margin of the bone as well as in the synovial membrane, are apparently induced by two factors—the irritative property of the foreign body and the faulty mechanics probably induced by its presence. That these causes must be at work is suggested by the experience with the Smith-Petersen arthroplasty, in which vitallium molds are inserted into the joint. For although the vitallium represents a foreign metal in the joint, it does not produce degenerative arthritis such as develops following accidental introduction of other types of foreign material. As a matter of fact, the presence of the vitallium cups in the Smith-Petersen arthroplasty results in a smooth joint surface, and the formation of rather healthy looking and acting fibrocartilage. Apparently this constructive (rather than degenerative) change induced by the vitallium cup is attributable to the fact that this metal is inert and that it is inserted into the joint in such a manner as to allow perfectly normal physiologic function.

NEUROTROPHIC FACTORS

The neuroarthropathies of tabes dorsalis, syringomyelia, and other lesions of the spinal cord, are essentially bizarre forms of hypertrophic arthritis. Fundamentally, they have a distinctive etiologic factor—the disorganization of certain neurogenic influences regulating joint function. However, the ultimate pathologic and clinical manifestations may be regarded simply as those of an ordinary hypertrophic arthritis resulting from postural trauma facilitated by loss of joint sensibility to pain and by relaxation of supporting periarthicular structures. This type of joint disease is to be discussed more specifically in a subsequent section (page 355).

METABOLIC FACTORS

Glandular disturbances in the direction of underfunction, particularly of the thyroid gland, may contribute to the progression of hypertrophic arthritis, either through the direct effect of decreasing thyroid secretion or, as is more likely, through slowing of the circulatory current that is one of its major effects. Over half of the patients with hypertrophic arthritis reveal fairly conclusive evidence of some degree of thyroid underactivity, many of these are distinctly improved by small doses of desiccated thyroid. Hypertrophic arthritis may be considerably aggravated by coexisting myxedema.

It has been implied that the glandular disturbance that is a sequela of the menopause is related to the production of hypertrophic arthritis. To be sure, such arthritic phenomena do become evident shortly after the menopause, thus suggesting some causal relationship. However, it is doubtful whether there is sufficient reason to attribute to any menopausal disturbance a *primary importance in the production of this form of joint disease*. The tendency to obesity, frequently associated with the menopause, may indirectly favor the development of hypertrophic arthritis, particularly in those persons who have already subjected their joints to some injury either from pre-existing obesity or from incipient senescent changes. In such cases, however, the menopause is merely another unfavorable incident in a chain of events favoring the production of hypertrophic arthritis.

It is tempting to consider the possibility of some error in general metabolism as a cause of the widespread degenerative changes of hypertrophic arthritis. Numerous hypotheses have been advanced concerning the "*metabolic*" basis of this condition, but these have not been substantiated by scientific investigation. As we have already mentioned, Bennett and Bauer showed conclusively that the hypertrophic arthritis experimentally in

duced in rabbits results from purely local mechanical disturbances, without intervention of any systemic or metabolic factor. Disturbances of metabolism in the usual sense of that phrase, cannot be said to exist as a primary cause of hypertrophic arthritis. We have already alluded to the contributory role of thyroid underfunction. The effect of obesity in hypertrophic arthritis is more mechanical than metabolic, and its genesis more often exogenous than glandular. Again, the theory that a 'calcium or parathyroid disturbance' is related to the arthritis is unsubstantiated by any proof whatever. Only if the sum total of metabolic processes constituting senescence and obesity be considered as the metabolic errors fundamental to hypertrophic arthritis may one assume that the disease is metabolic in nature.

The frequent occurrence of disturbances in carbohydrate metabolism (sometimes frank diabetes) among patients with hypertrophic arthritis is readily explained by the greater incidence of obesity among these patients than among normal control groups. There is no indication, however, that such disturbances in carbohydrate metabolism have any relation either to the development of hypertrophic arthritis or to its usual symptoms. True, the neuritic pains associated with hypertrophic arthritis are greatly intensified by coexisting diabetes and are ameliorated by its control, but elimination of hyperglycemia alone does not suffice for complete control of these neuritic pains nor for prevention of their recurrence. Even when the diabetes is adequately controlled the arthritis requires direct treatment.

INFECTION

We have ample proof that infection probably plays no part in the production of the primary changes of hypertrophic arthritis. Here we need only recall those indicators of infection that are almost invariably present in atrophic arthritis and that are strikingly absent in hypertrophic arthritis. An increased rate of sedimentation of erythrocytes, a pronounced increase in the proportion of young nonfilamented polymorphonuclear leucocytes, the presence of streptococcal agglutinins and precipitins in the serum, and the clinical signs of systemic infection. However, the changes in the joints induced by senescence, trauma, obesity, or poor posture, undoubtedly make the joints more vulnerable to superimposed infection. For this reason atrophic arthritis may be superimposed upon and produce the greatest discomfort in weight bearing joints of the obese individual, and the treatment of such patients demands attention to the factor of infection as well as to the mechanical injury to the joints from obesity.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527]

Metabolic and Endocrine Background (Menopausal Arthritis)

- CECIL, R L, and ARCHER, B H Arthritis of the menopause A study of fifty cases *JAMA*, 84 75, 1925

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CHAPTER XVIII

PATHOLOGY OF HYPERTROPHIC ARTHRITIS

The earliest pathologic changes in hypertrophic arthritis develop in the cartilage, and not in the synovia, as in atrophic arthritis. The cartilage loses its normal lustre and develops a cracking or fibrillation of its matrix. As superficial layers of the cartilage crack, bits of it partially break away. In microscopic sections such degenerated cartilage resembles a tattered flag (Fig 95).

A progressive disorganization in the arrangement and size of the cartilage cells develops. With degeneration and peeling of surface layers of the cartilage plates they tend to assume an irregular outline, thinned in some places and pitted in others. The degree and extent of cartilage degeneration and fragmentation apparently depend upon the extent to which these areas are subjected to stress from trauma. The greater the degeneration of one portion of the cartilage the greater the stress to which the remainder is subjected and, therefore, the more pronounced the eventual pathologic picture. In time the entire thickness of cartilage may be degenerated so that the underlying bone is actually laid bare. It should be remembered that such erosion of cartilage results from primary degenerative changes and not from encroachment upon it by an inflammatory synovial pannus, as occurs in atrophic arthritis.

As the superficial layers of the cartilage are degenerating and desquamating its deeper layers, those immediately adjacent to the subchondral plate of bone, undergo increasing condensation and calcification. By the time a considerable thickness of the cartilage has been destroyed, the subjacent bone has become hardened, eburnated. This change is apparently stimulated by mechanical stress of function when the integrity of the cartilage has been impaired. The new joint surface is then represented by an irregular plate of dense, eburnated bone being either the calcified basal layers of the former cartilage plate or, if that has been destroyed, the subchondral epiphyseal bone.

The bone trabeculae also become thicker and harder, sometimes coalescing and obliterating the marrow spaces which formerly intervened between them. Except for such encroachment the marrow itself does not participate in the pathologic process. Inflammatory foci do not develop in

the marrow as they do in atrophic arthritis. It is evident, then, that in the subchondral epiphyseal bone the pathologic changes in hypertrophic arthritis are practically the antithesis of those we have noted in atrophic



FIG. 9. Marked degeneration of cartilage, with fibrillation and fragmentation of its surface layers (From R. K. Ghormley, *The Pathology of Non Specific Arthritis*, In *A Survey of Chronic Rheumatic Diseases* Oxford Press 1938.)

arthritis. In the latter, decalcification of subchondral bone occurs, the trabeculae become thinner, less dense and the marrow spaces proportionately larger.

At the joint margin, along the line of reflection of the synovia onto the perichondrium, the pathologic change assumes a somewhat different character. This area, unlike the cartilage plate, is composed of a rather vascular fibrous tissue. Because of this fact, the pathologic reaction along this line is primarily one of hyperplasia. Apparently because irregularity in the contour of the joint's surface induces excessive mechanical stress, this hyperplastic ring of fibrous tissue undergoes metaplasia with gradual transformation into cartilage and, eventually, into bone. This margin of proliferating bone is made up of irregular osteophytes, or spurs. These represent the most

obtrusive, gross pathologic change of hypertrophic arthritis, so readily apparent in roentgenograms. Like the margin of a coral reef these osteophytes practically encircle the joint surface at the ends of the bone.

Though fragmentation of cartilage, its gradual erosion, eburnation of the underlying cartilage and bone, and marginal hypertrophic spurs represent the primary and characteristic pathologic changes of hypertrophic arthritis, the synovia also becomes implicated. The synovial membrane is stimulated to hyperplasia of its lining cells, and particularly of its villi. This hyperplasia may be induced by trauma of mechanical pressure from abutting hypertrophic spurs along the margin of the joints and is probably intensified by trauma of weight bearing and joint function. For this reason, it is particularly marked when trauma from abnormal alignment at the joints, resulting from static abnormalities, coexists. In the knee these masses of thickened synovial tissue are generally most easily palpable along its mesial surface.

The hyperplastic synovial membrane and villi may undergo further changes. Again as a result of continued trauma, sections of the synovia, particularly its villi, may be stimulated to metaplasia into cartilage and even into bone, constituting essentially masses of osteochondromata. These may be torn from their pedicles, becoming loose "joint mice," lying free within the joint cavity. Time and friction may wear the surfaces of these loose osteocartilaginous bodies quite smooth and hard. They may insinuate themselves between the surfaces of the joint in such a manner as to produce pain or mechanical locking of the joint.

Significantly, such synovial hyperplasia (villous synovitis) is devoid of cellular infiltration and vascular congestion, such as occurs so characteristically in atrophic arthritis. Because the synovia and cartilage are devoid of an inflammatory reaction there is practically no tendency to ankylosis. However, extensive alteration in the architecture of the joint may result in serious interference with its function. Interlocking of marginal osteophytes or obstruction from masses of thickened synovia or loose bodies may make full flexion or extension of the joints impossible and may induce some deformity. Although it is seldom of severe grade, long persisting flexion deformity may lead to contracture of the joint capsule and, eventually, to inability to extend the joint fully. This practically never results from fibrous or bony union.

The synovial fluid is seldom increased, it is frequently considerably decreased in amount. Small effusions may develop if acute trauma is inflicted upon joints affected by hypertrophic arthritis. However, these effusions do not persist, being readily absorbed when the joint is put at rest. The capsule may undergo concomitant degenerative changes, developing fragmentation

of its fibrous tissue minute hemorrhages and subsequent calcification. But again inflammatory changes with cellular infiltration do not appear. Muscle atrophy too so evident in atrophic arthritis is absent in hypertrophic arthritis except when disuse of a joint induces wasting of related muscles.

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CHAPTER XIX

CLINICAL MANIFESTATIONS OF HYPERTROPHIC ARTHRITIS

Hypertrophic arthritis may be entirely latent and produce no symptoms. When it does produce disturbing manifestations, their severity is determined not only by the degree of pathologic change and by the aggravating influences that may exist, but also by the patient's sensitiveness to pain.

The symptoms of hypertrophic arthritis generally creep in insidiously, usually in middle age, but often the actual pathologic change had begun many years previously. We have already indicated that this condition is not confined to those of middle age, symptoms may appear before the age of forty, even in the twenties.

Except for the much greater susceptibility of women to Heberden's nodes, hypertrophic arthritis affects men just about as often as women. The contributory influence of the menopause is fairly well balanced by the effect of more severe trauma to which men are exposed. Symptomatically, however, the effect of the menopause is apt to be more evident.

Because trauma plays such an important role in inducing the pathologic changes of hypertrophic arthritis, the condition is generally seen in its most striking form in manual laborers, farmers, housewives, and obese persons. But the symptoms of the disease are not necessarily proportional to the degree of pathologic change. The coal miner with pronounced and extensive hypertrophic arthritis in his spine and knees may be less uncomfortable than the sensitive matron with a few Heberden's nodes.

We have already stated that trauma aggravating hypertrophic arthritis may arise from obesity, accidents, occupational, and even recreational pursuits. The effect of severe trauma on pre-existing hypertrophic arthritis is brought out pointedly by abrupt exacerbations in the arthritic process after accidental falls or injuries. Under such circumstances previously symptomless hypertrophic arthritic changes may flare into progressive activity, with severe pain and disability. Though equally severe injuries to other joints may end in complete recovery, injuries inflicted upon a joint fundamentally prepared for, or already the seat of, hypertrophic arthritis may lead to disastrous damage. Exacerbation in the activity of this condition is not, however, a universal sequela of trauma. The author has observed the develop-

ment of stubbornly persisting pain and activity in cases of previously quiescent hypertrophic arthritis subsequent to overzealous massage or exercise, and on the other hand, he has seen cases of this type of arthritis, which were unharmed after relatively severe traumatic injuries. The relation of physical trauma to the onset of symptoms and disability in patients with hypertrophic arthritis can usually be determined with accuracy, however, through competent analysis of all the facts obtained through careful medical study.

Because of the effect of trauma in precipitating symptoms, the joints which are apt to be the most painful are those of the cervical and lumbar spine, the knees, and the terminal joints of the fingers. The hip joints are next in order. The proximal phalangeal and the metacarpophalangeal joints of the hands and the wrists are rarely affected. The shoulders, also, are an infrequent site of hypertrophic arthritis with symptoms.

A patient presenting evidence of hypertrophic arthritis in many joints may complain of pain confined to one or two of them, those which are subjected to traumatic strain. Those free of such strain are symptomless. Even joints with most glaring hypertrophic change may be ignored by the patient because they are relatively painless, whereas other joints with only the slightest grade of visible pathologic change may cause considerable discomfort. The large hypertrophic knee may be painless while the opposite, relatively normal appearing one may be quite sore on motion. Unlike the distribution of the process in atrophic arthritis, that in hypertrophic arthritis is generally asymmetrical.

The common manifestation is aching discomfort, particularly during use of the joint or shortly after. There is generally little pain on passive activity through the available arc of motion, and limitation of motion is rarely noted, or only of slight degree.

The pain of hypertrophic arthritis is generally mild, it is likely to occur intermittently at first and to be aggravated by activity. Going up and down steps is particularly painful, if the knees are affected. Episodes of subacute or even acute pain may supervene. They are generally brought on either by acute accidental trauma or by microtrauma from overuse of the joints, frequently under the disadvantage of poor body mechanics. The author has seen many instances in which the acute symptoms were brought on inadvertently by the patient's misdirected efforts to relieve his milder symptoms by vigorous exercise. Some of these patients confine their treatment to 'setting up' exercises, but others adopt violent exercises in the hope of retaining as much suppleness of the joints as possible. Driven by the fear of ankylosis which, we repeat, practically never occurs in this disease, some patients persevere with such activities despite pain, and sometimes, indeed add to the amount of exercise with increase in pain.

The symptoms of hypertrophic arthritis may be brought on by excessive indulgence in sports requiring much physical effort. Spurred on by a subconscious effort to prove to himself that he is not as old as he actually is the middle aged or elderly patient may pursue such activities with excessive zeal and consequent injury to the joints. It is important to inquire about these factors for not realizing the relationship the patient is not likely to volunteer the information that is so necessary in this connection.

In addition to various types of trauma it is possible that acute infections of various sorts localize at times in affected joints and produce an inflammatory process with aggravation of formerly mild symptoms.

In addition to pain these patients may complain of stiffness after rest relievable by limbering up on walking. Such stiffness is probably referable to associated periarthritic degenerative changes in the capsule and fibrositic changes in the muscle.

Physical and mental fatigue is often an associated symptom. Even when it is disturbing the patient may not complain of it unless he is questioned specifically on the matter. His immediate concern is about the joint pains and he may not be consciously aware of the fact that the onset of joint disability followed immediately upon a period of prolonged nervous strain induced by worry or anxiety.

Naturally patients may present symptoms concomitant with the arthritis but not directly related to it. Insomnia, vague digestive disturbances, constipation and paresthesias are not infrequent accompaniments. The paresthesias may be aggravated by coexisting anemia, either hypochromic or macrocytic in type. These patients may be aware that they 'puff' on climbing a hill or on going up steps, either because they are overweight or because they have associated hypertension and myocardial insufficiency. The vasomotor and nervous instability of the menopause is evident in some of them.

Aside from the joints the patient with hypertrophic arthritis may reveal few abnormal physical signs. In fact he generally appears quite healthy, sometimes extraordinarily robust, large framed and nourished to excess. This is in contrast to the drawn, pallid or pasty complexioned patient suffering from atrophic arthritis. The temperature is normal, there is usually no tachycardia. Sometimes there is slight cardiac enlargement associated with moderate or more severe hypertension.

The course of hypertrophic arthritis is generally benign. Ankylosis practically never occurs. Deformity from muscular contractures develops rarely. For muscle spasm of severe degree is seldom induced. If adequate treatment is applied early the degenerative process in the cartilage and the secondary hypertrophic changes in the bone may be arrested and functional usefulness of the joint preserved despite the fact that restoration of its anatomic

integrity is impossible. Only if the disease is permitted to progress unchecked does one observe disabling alteration of the joint, through the formation of extensive osteophytes which limit the range of motion and increase the degree of disability through pain. Even then the joint is seldom completely useless, pain resulting only from the trauma of exercise and abating or disappearing when the joint is at rest. To be sure, the osteophytes projecting from the margins of adjacent vertebrae may coalesce slightly impairing the normal flexibility of the spine, but the patient is then rewarded by spontaneous relief from pain, the relief being attributable to the buttressing effect such fusion brings about. Altogether, the natural course of hypertrophic arthritis, when uncomplicated by infection or accidental injury, reveals its strikingly innocent character. Serious damage from it need not generally be feared.

LABORATORY FINDINGS

There is striking difference between the results of laboratory studies in hypertrophic and atrophic arthritis. In hypertrophic arthritis the total leucocyte, erythrocyte, and differential counts are normal. In the majority of cases the usual proportion of nonfilamented polymorphonuclear leucocytes is not increased. The sedimentation rate is normal (Fig 96), as are the urine and chemical studies of the blood. Coincidental anemia, diabetes, cardiorenal disease, or arteriosclerosis may modify the laboratory findings, but hypertrophic arthritis alone produces practically no abnormalities that can be found in the course of clinical laboratory examination.

ROENTGENOGRAPHIC MANIFESTATIONS

Except in the earliest stages of the disease, when the degree of cartilage degeneration is too slight to be revealed in the x ray, the roentgenographic appearance of the joints in hypertrophic arthritis is characteristic. With progressive degeneration of cartilage there is, of course, narrowing of the joint space, as in atrophic arthritis, but in addition, there are features characteristic of hypertrophic arthritis only. Instead of the diffuse osteoporosis about affected joints, the bone retains its radiopacity. In fact, the bone just beneath the affected joint cartilage may appear more dense than normal. The margins of the joints are dotted with pointed or rounded overgrowths, constituting the characteristic osteophytes of hypertrophic arthritis. In the knees, loose bodies may be present in association with other hypertrophic arthritic changes (Fig 97). Even when the cartilage is entirely destroyed, evidence of bony ankylosis is absent, although osteophytes projecting from

the margins of the vertebrae do sometimes, produce bony bridges which unite the vertebral bodies

Hypertrophic arthritis is prone to affect certain joints more than others

BLOOD SEDIMENTATION TEST

Case No. 1055

Name. Mrs L F

Diagnosis. HYPERTROPHIC (OSTEO) ARTHRITIS

Date. February 25 ____ 1938

Tube No. 1

Readings by M F

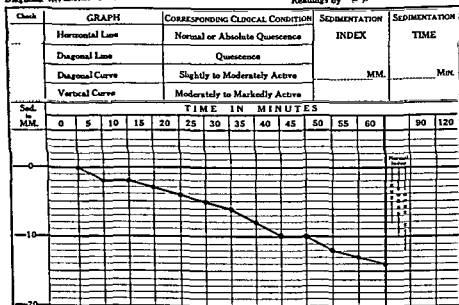


Fig 96 Diagonal line indicating essentially normal sedimentation of erythrocytes in a case of hypertrophic (osteo) arthritis

producing characteristic clinical pictures These will be discussed in the following pages

HEBERDEN'S NODULES

In the hands the most distinctive lesions of hypertrophic arthritis are the hard irregular knobs over the distal phalangeal joints As long ago as 1802 Heberden recognized and described these nodules as manifestations of a relatively innocuous type of arthritis which incidentally he realized was not gout Women are more subject to Heberden's nodes than men The only obvious indication of hypertrophic arthritis may be these nodules of the fingers although further search in such cases usually reveals symptomless hypertrophic arthritic lesions in other joints as well These nodes apparently represent proliferations of the synovial membrane and capsule over the dorsum of the distal phalangeal joints Degeneration of the car

tilage and some degree of hypertrophic arthritic change at the margins of the distal phalanges are frequently associated

Heberden's nodes present such a characteristic appearance that they



FIG. 97 Hypertrophic (osteo-) arthritis of the knee with osteophytes, loose bodies and slight effusion. Note also the narrowed joint space resulting from cartilage degeneration and the increased density of the bone in the subchondral zone. (From Ferguson, Roentgen Diagnosis of the Extremities and Spine, Paul B. Hoeber, Inc., 1939.)

may be recognized at a glance (Fig. 98). They occur only at the distal finger joints and may be present either on only one or two fingers or on most of them. These nodes may stand out as the only abnormality in the hands, for the proximal phalangeal joints appear normal. When Heberden's

nodes develop slowly and painlessly the patient accepts them without much concern. Elderly people particularly are likely to recognize these knobs as a manifestation of their age which they actually are. Not inconvenienced

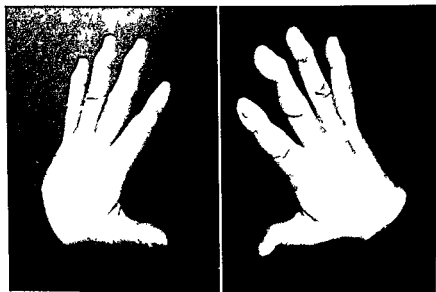


FIG. 98. Heberden's nodes at the distal phalangeal joints, the typical manifestation of hypertrophic (osteo) arthritis in the hands.

by their presence they never seek medical advice on account of them alone. Younger patients and women particularly are likely to be more concerned about their development. Discomfort or impairment of joint function is less apt to bring such a patient to the physician than the fear instilled perhaps by a well-meaning friend that this may be the beginning of a crippling arthritis.

When Heberden's nodes do cause symptoms they are likely to appear during the early stages of development of the nodes. There is never severe pain; there is rather a sensation of soreness, particularly when using the fingers or on lateral pressure. When the patient complains of soreness and swelling there is likely to be some tenderness on pressure over the nodules and the skin may be somewhat red and glossy. Such features suggest to the patient the possibility of an inflammatory process and they cause increasing apprehension. Heberden's nodes may become sore and tender after overuse of the fingers, either from exercises carried out with the deliberate aim of keeping the joints from getting stiff or from excessive use. Heberden's nodes rarely impair motion at the joints to any significant extent. In time these nodes attain maximum development (never to alarming proportions) pain or soreness, if it has been present, becomes negligible; the

deformity alone may then be the most disturbing aspect to the sensitive patient

THE KNEES

The knees are frequently involved in hypertrophic arthritis and are likely to cause distress sooner or later. This is due to their exposure to physical strain from physiologic use, from static defects of the feet, and from obesity.

The initial subjective manifestation of hypertrophic arthritis in the knees is a dull aching, aggravated by walking, particularly up and down steps, descending stairs is more difficult and painful than ascending them. The aching is apt to be most pronounced toward the end of the day, or after a period of exercise. On the other hand, there may be considerable stiffness when the patient attempts to walk immediately after sitting a while. However, the pain becomes severe only if the joint is subjected to excessive trauma.

Often the patient complains of creaking in the knees. Swelling, if it appears at all, is slight or only moderate in degree, and is altogether unlike the swelling in atrophic arthritis, there is no boggy periarticular swelling (Fig 99). In patients with long neglected static defects of the feet or with knock knees, the mesial aspect of the knees may be visibly enlarged. On palpation a large mass of rather firm tissue, representing the markedly hypertrophied synovial membrane, may be found. Except for such thickening caused by synovial hyperplasia, the enlargement of the knee affected by hypertrophic arthritis yields a sensation of bony hardness—a somewhat irregular, knobby, bony hyperplasia.

Motion may be free, except when the joint becomes locked by loose joint bodies, or, in more acute cases, when complete extension is limited by spasm of the hamstring muscles. Tenderness may be elicited over the lateral margins of the joints, but pain and tenderness are not proportionate to the extent of the pathologic change evident. A large knee with obviously marked hypertrophic change may be relatively painless, both on passive and active motion, whereas the other knee, showing very little change from normal, may be quite sore on motion. Creaking is usually clearly audible, sometimes a sensation of gritty crepitus is imparted to the palpating hand. Passive movement through the available arc of motion may be entirely painless, particularly if the joint is moved slowly, gently. Less guarded motion may induce sudden spasm of muscles and pain. Synovial effusions are rare and there is, as a rule, no muscle atrophy.

When loose cartilaginous bodies—"joint mice"—are present, they may insinuate themselves between the surfaces of the joints, producing sudden

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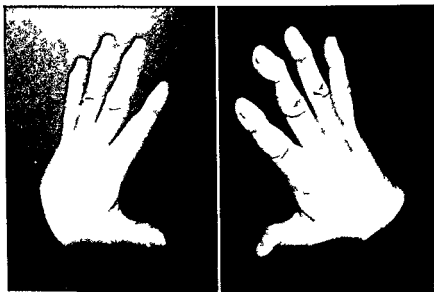


FIG. 98 Heberden's nodes at the distal phalangeal joints: the typical manifestation of hypertrophic (osteo-) arthritis in the hands

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When loose cartilaginous bodies—"joint mice"—are present, they may insinuate themselves between the surfaces of the joints, producing sudden

pain and muscle spasm causing the patient to halt. Active manipulation or rest may bring temporary relief but unless these cartilaginous fragments are removed episodes of pain are likely to recur without warning.



Fig. 39. Hypertrophic (osteo-) arthritis affecting the knees and other weight bearing joints. Note the obesity and pronation of the feet.

THE HIP

Hypertrophic arthritis of the hip (*malum coxae senilis*) may occur independently of other hypertrophic arthritic lesions and it generally occurs during early or late middle life. Obviously, the condition is seen in its most pronounced form when the patient reaches an advanced age. It is particularly likely to develop in patients who are obese and in those who for many years have subjected the hip to postural strain from static malalignment at the feet or knees. The condition may begin in one hip and involve the other at a subsequent time. In most cases both hips are eventually affected although the involvement in one may be greater than in the other. Hypertrophic arthritis of the hips may be extremely disabling.

The pathologic changes and roentgenographic features are characteristic. The head of the femur is ground down and flattened, the acetabulum is widened and its margin becomes dotted by a fringe of irregular, hypertrophic spurs. As the head of the femur is worn down, the joint space becomes proportionately narrowed; it may disappear entirely. There is usually, in addition, hyperplasia and thickening of the synovia, but that is not likely to be readily perceptible clinically, because the joint is so deep seated.

Pain is generally induced by motion, ordinarily by walking, so that the degree of discomfort varies with the amount of use. The patient is also conscious of stiffness in the hip, caused by muscle spasm. Sometimes the pain radiates to the groin or to the knees, as is not uncommon in all types of hip joint disease. Examination reveals limitation of rotation, later, limitation of abduction and adduction, with pain at the extremes of these motions. Finally, even flexion and extension of the hip may become drastically limited and painful. If marked destruction of the femoral head occurs, actual shortening of the leg, exaggerated by adductor spasm, may develop. The gait is then disturbed both by pain and limitation of hip motion. Secondary changes in the lumbar curve may be induced.

THE SHOULDER

Although audible creaking is commonly elicited on passive or active motion at the shoulder, particularly in patients over forty years old, pain in this joint is seldom caused by hypertrophic arthritis alone. The discomfort is usually the result of associated periarthritis or subacromial bursitis. Yet the author has encountered rare instances in which the head of the humerus as well as the glenoid fossa and the acromioclavicular joint, was affected by extremely marked hypertrophic changes which produced not only a coarse, grating noise on motion, but also pain, evidently resulting from irritation of the synovia. In such cases there was usually limitation of motion attributable directly to mechanical interference by the extraordinarily large osteophytes.

THE SPINE

The spine is probably the most common site of hypertrophic arthritic changes. The pathologic reaction here is essentially the same as in other joints, developing at the margins of the vertebral bodies and at the lateral intervertebral joints. Pain is not, however, a constant accompaniment, even when this type of spinal arthritis exists pathologically. It has been shown, for example, that arthritic changes in the lower spine can be detected by roentgenograms in 60 to 70 per cent of all persons aged over fifty years,

although the vast majority of them are not in the least aware of the condition. The lumbar spine is most commonly affected, the cervical segments next often, and the dorsal spine next.

CERVICAL SPINE

Hypertrophic arthritis of the cervical spine may produce a great variety of symptoms depending on which of the cervical vertebrae are affected and on whether the symptoms result directly from arthritic change in the vertebrae, involvement of the ligaments, or whether they result from nerve root irritation. There may be only stiffness or a constant aching confined to the back of the neck and base of the head, frequently associated with a dull morning headache. Creaking on motion may be very disturbing even when not painful. Stiff neck may appear intermittently, causing pain for a day or two, then clearing spontaneously or after a dose of aspirin or the application of heat. The pains may be more severe; they may be sharp, radicular in character, radiating over the occiput to the ear, over the neck, or across the posterior aspect of the shoulders and down the arms. The root pains which may result from cervical hypertrophic arthritis are discussed more fully in another section (page 399).

Although the patient may attribute the onset of his symptoms to drafts or inclement weather, it will usually become apparent from the history that these manifestations appeared shortly after a period of either excessive nervous or physical strain. Having so often seen acute symptoms of cervical hypertrophic arthritis precipitated by an acute emotional upset associated with severe nervous and muscular tension, the author is convinced of the relationship between them. Postural strain, such as may occur during a long automobile drive or from work requiring flexing the head for long stretches daily, may induce such symptoms.

Examination reveals considerable stiffness of the neck, resulting from muscle spasm. It is most evident on rotation and lateral flexion of the head, not so much on forward and backward bending. Palpation of the posterior nuchal muscles may reveal tenderness and induration of the periarticular structures.

Headache is a common manifestation of cervical hypertrophic arthritis, particularly when there is associated fibrositis. Although such headache is frequently associated with frank arthritic changes, it is probable that the symptoms are really induced by fibrositis affecting various ligaments and muscles about the cervical spine and occiput. The headache may be erroneously attributed to other etiologic factors. Careful analysis of the history aids in correlating the symptoms with the existing arthritis, however.

Such headache is generally described as beginning in the occiput, then

spreading forward to the vertex or temples. It generally begins early in the morning, sometimes awakening the patient. A sensation of stiffness and soreness in the upper cervical region may be present and tenderness may be elicited.

Such headache may occur intermittently at first, each bout lasting one to four days, with the diurnal variations in severity already described, namely, exaggeration of pain in the early morning hours, followed by abatement toward the latter part of the day. Bending the head forward may aggravate the headache. Temporary relief may be secured by bending the head backward, by heat and massage or by taking salicylates. The condition may become so constant and nerve wracking that it amounts to serious disability. Such patients are literally harassed by a feeling of tightness and stiffness in the back of the head and neck which they describe as a sensation of "fullness and congestion," or as a constant, dull pain.

A creaking sensation, extremely annoying and disturbing to the patient when he turns his head, may also be noted. The creaking may be audible even to the examiner, or he may just sense a characteristic crepitus, as he rotates or moves the patient's head laterally.

The character of the headache, its situation, the circumstances under which it appears, and the absence of nausea and vomiting, exclude migraine. Moreover, such headaches do not usually make their appearance until middle life, nor is there a familial history of migraine. Evidence of one type of rheumatic manifestation or another, in situations other than the cervical spine, may be obtained, such information helping to confirm the diagnosis of "rheumatic headache."

In addition to degenerative changes in tendons and muscles and to frank hypertrophic arthritis in the spine, other factors may contribute to the production of the symptoms described.

Focal infection is unquestionably one such aggravating influence, and is most likely to be manifest in younger persons. Debility from physical or nervous strain, unfavorable atmospheric or climatic conditions, various grades of hypothyroidism, anemia, postural strain—any or all these may facilitate the production of such symptoms or aggravate them. It is questionable whether dietary indiscretions, particularly excesses in starches and sweets, or colonic stasis, contribute much to this clinical syndrome. A clear cut allergic relationship is also rarely noted. Although ocular disturbances, conducive to postural strain, may aggravate the headaches and may therefore require correction, it is important not to ascribe the headaches to a purely ocular basis.

The diagnosis depends chiefly on eliciting and interpreting the relevant details of the history. Evidence of cervical hypertrophic arthritis in roentgenograms adds confirmatory proof, but such findings alone do not estab-

lish a diagnosis nor does their absence exclude the existence of this condition

THE DORSAL SPINE

In addition to producing discomfort directly over the dorsal region of the back hypertrophic arthritis of the dorsal spine may project pain along the course of distribution of related dorsal nerve roots. Thus, pain arising in the upper dorsal spine may radiate anywhere along the back of the chest or forward to the pectoral region. If the pain is over the left pectoral region or sternum as it may be the possibility of heart disease may present itself to the patient as well as to the physician. The fact that pain is produced by activity such as walking bending turning and that it may be relieved by lying down may strengthen the suspicion. Closer analysis reveals however that in addition to discomfort over the sternal or pectoral regions the pain radiates along the ribs to the back. A burning pain quite unlike the oppression produced by angina pectoris may be associated and such pain is relieved by the use of heat or aspirin.

The discomfort of dorsal hypertrophic arthritis may be felt lateral to the spine in the interscapular regions suggesting pleurisy. Root pains arising from the lower dorsal spine may radiate along the lower rib margin, suggesting cholecystitis or some other abdominal visceral condition. From the lowermost portion of the dorsal spine pain may radiate to the lower abdomen suggesting the possibility of appendicitis ureteral disease of colitis. Unnecessary operations may be performed to eradicate presumed abdominal visceral disease. Some of these patients have undergone a second operation for presumed adhesions when as a matter of fact more careful study in the beginning might have revealed the real nature of the condition.

THE LUMBAR SPINE

Hypertrophic arthritis of the lumbar region of the spine may produce symptoms either confined to the lower back or projected along the course of distribution of the roots of the sciatic nerve. This subject will be discussed in detail later in a consideration of low back and sciatic pain (page 457).

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 52-]

CHAPTER XX

TREATMENT OF HYPERTROPHIC ARTHRITIS

Many patients with hypertrophic arthritis never seek treatment for it because they either suffer no pain at all or only a mild degree of discomfort, which is not distracting enough to annoy them seriously. Every physician has encountered elderly patients whose knees creak and whose hands indicate the signs of advanced hypertrophic arthritis. Yet these patients are as oblivious to the condition of their joints as they are to their wrinkles and grey hair. They regard their round knobbed fingers imperturbably as something to which they have fallen heir, something which was their due. And they look with a certain naive surprise at a physician who, seeing the patient for the first time, may exhibit some passive curiosity about these changes in the joints.

Other patients go to the physician because of anxiety over their future. They know the devastating effects of certain cases of arthritis and are panic stricken lest they, too, be destined for the same end. Fear of crippling and invalidism motivates their visit to the doctor. *Perhaps the most important service the physician can render these patients is to allay such apprehension. It must be pointed out to them that deformity and ankylosis of such joints practically never occur. Such reassurance is all some patients ask. It is important even when further treatment is necessary, for it allays anxiety.*

Still other patients with Heberden's nodes, oversensitive to the slight irregularity of the fingers, may seek a means of ridding themselves of this deformity. They must be convinced that the changes in the terminal joints are irreversible. Once they realize that this slight disfigurement is inevitably permanent, they usually accept it philosophically.

Much can be done in preventing the symptoms of hypertrophic arthritis from appearing, even though the pathologic changes of this condition already exist. In such a case the patient must be advised against permitting himself to become obese, he must recognize the necessity for avoiding excessive strain on the joints. Some of these patients must be reminded that they are not as young as they were, that they cannot comfortably indulge in their customary physical activity or sports. On the contrary, they must provide for more rest, longer hours of sleep, and even rest periods

during the day. Postural errors particularly of the feet should be corrected before they produce symptoms of strain on the joints. Sometimes it is well to protect vulnerable joints against undue strain by means of bandages, braces or other supports.

When the patient complains of discomfort its precipitating cause must be sought and if possible eliminated. Exercises, vigorous massage, are not only unnecessary but frequently harmful for the joints are more likely to be injured than helped in this way. Relief from trauma or undue physical strain is more logically indicated. The joints particularly in weight bearing regions are greatly helped by rest judiciously applied. The patient need not be converted into an invalid. Moderation is essential. Measures of general hygiene including adequate rest periods and relaxation and a well balanced regimen of living to diminish the wear and tear at this period of life are obviously of additional help.

The condition in the joints is ameliorated by the usual methods of physiotherapy. Assuming that a decrease in circulation to the joint tissues is a factor in the production of hypertrophic arthritis heat should prove beneficial. By increasing the circulation in this manner one initiates optimum conditions for repair. Physiotherapy sporadically applied once or twice a week, is no more than a therapeutic gesture; it should be available once or twice daily and it is most likely to be so employed if the patient has a baker at home. A simple radiant heat lamp can be rigged up at little expense as indicated on page 232.

The contrast bath and hot paraffin applications (page 233) are valuable in the treatment of Heberden's nodes. The simplicity of these methods enhances their value though some patients prefer a more elaborate setting for therapy. The blood vessel exercise effected by the contrast bath results in significant improvement in the function of the remaining available circulation.

The details of the physical therapeutic management in chronic arthritis have already been discussed (page 231).

Drugs for relief of pain are rarely necessary in this form of arthritis. Relief through rest and physiotherapy is often prompt. Without resort to these measures drugs are mere palliatives. Acetylsalicylic acid is a simple yet effective analgesic which may be employed if joint pains persist despite the institution of rest particularly if the discomfort disturbs sleep.

Thyroid extract may be useful; it is frequently extremely helpful when there is indication of a lowered rate of metabolism or frank myxedema.

The existence of associated anemia dictates the necessity for its control. Even mild degrees of hypochromic anemia should be corrected by the administration of iron in adequate amounts. Liver extract is generally unnecessary unless there is frank hyperchromic anemia. Vitamins should be

employed only if there is clear indication of vitamin deficiency Dilute hydrochloric acid may be administered if there is associated achlorhydria, as there may be, in patients of advanced age Estrogenic substance should be used only when there is need for controlling disturbing menopausal symptoms

Since infection apparently does not play any part in the process, eradication of foci of infection will not influence the course of the disease Obvious foci of infection may be removed however, on principles of general hygiene or to prevent superimposed infection of handicapped joints Vaccines are not logically employed in this type of arthritis

When hypertrophic arthritis is aggravated by the mechanical strain of overweight, the obesity must be eliminated by reduction in the caloric intake of food Except for such dietary restriction, no special modification of diet is required Reducing the amount of protein particularly of meats is not only unnecessary, but actually unwise There is no indication of a relationship between the protein intake, or the kind of protein eaten, and hypertrophic arthritis

Postural deformities in the feet and ankles, with shifting of the load of the body weight to unusual situations, may be the cause of persisting disability when every other factor has been excluded Such static defects in the feet must be treated by fitting with appropriate shoes Frequently the shoes must be altered specifically to correct the deformities peculiar to the individual patient

Mechanical supports such as corsets, properly fitted "orthopedic belts" and braces, roller bandages, and foot supports, may add stability and reduce discomfort when the patient is active

TREATMENT OF MENOPAUSAL ARTHRITIS

Although we do not subscribe to the view that a specific hormonal disturbance, characteristic of the menopause, is primarily related to the production of so-called 'menopausal' hypertrophic arthritis, we feel certain that the arthralgia of this disease may be aggravated, to an extent at least, by the menopause Whether there is merely generally heightened sensitivity of the nervous system to pain, or whether other factors inherent in the menopausal state are responsible, is of no importance at the moment Clinical experience indicates, however, that such patients respond satisfactorily to the administration of estrogenic hormone, with amelioration of symptoms and sometimes dramatic subjective relief When the use of estrogenic hormone appears indicated, it should be provided in adequate dosage Treatment may be started with 5,000 to 10,000 rat units given parenterally,

once a week for from four to eight weeks followed by smaller doses by mouth for a more extended period. The injection of massive doses of estrogenic hormone over long periods of time may not be devoid of certain undesirable effects. Such dosage has been suspected of producing ovarian cysts, cystic mastitis and perhaps even carcinomatous change in the breast.

PREVENTION AND TREATMENT OF DEFORMITIES

The principles that govern the prevention and treatment of deformities in chronic arthritis have already been discussed (page 167). The same principles that apply to the care of the joints in atrophic arthritis apply also to those in hypertrophic (osteo-) arthritis. There are however variations in detail dictated by the differences in the pathologic process in each of these types of rheumatic disease.

We have emphasized that the pathologic changes in hypertrophic arthritis are of a bland noninflammatory type. Hyperplasia of the synovial membrane and its villi may develop but it is quite different from the inflammatory pannus of atrophic arthritis. Cartilage degeneration occurs but the hypertrophic bony changes are the more prominent end result. Unlike the situation in atrophic arthritis muscle spasm is a negligible factor not seriously conducive to producing deformity. Muscle atrophy does not occur except to that slight degree which inactivity forced by pain may induce. Hence the prevention of deformity in hypertrophic arthritis is really a simple matter. The natural tendency is for it not to occur at all.

Surgical Measures

When deformity and limitation of mobility do occur they are largely the result of mechanical interference with joint function either from hypertrophic spurs, loose cartilaginous bodies, excessively thick hyperplastic synovia or pain.

Surgical measures are necessary in those advanced cases in which extensive and permanent pathologic change has introduced deformity or interference with the usefulness of a joint. Synovectomy may be very helpful by restoring the normal range of mobility and by eliminating pain in cases of villous synovitis in which extreme synovial hypertrophy causes mechanical interference with smooth and normal function and in which resolution cannot be effected by rest and physiotherapy. Arthrotomy for removal of loose cartilaginous bodies or for excision of large exostoses with or without removal of the patella may be necessary in those cases in which repeated locking of the joint occurs or in which extensive hypertrophic bony change interferes with the desired degree of flexion or extension.

The results of such operations are generally extremely rewarding. Not only may all or most of the normal range of function be restored, but relief from pain may be obtained at the same time. Contributory factors such as obesity, static defects in the feet, and so on, must of course be eliminated or corrected simultaneously if recurrence is to be obviated.

In the treatment of hypertrophic arthritis of the hip (*malum coxae senilis*) we have found conservative measures disappointing. Drilling of the femoral epiphysis (MacKenzie-Henderson) has also failed in our limited experience with this operation. Surgical arthrodesis of the hip has in the past been the only therapeutic measure certain to relieve pain. This operation is applicable to cases with unilateral involvement of the hip. The more recent introduction of the Smith-Petersen arthroplasty technique, interposing a vitallium cup between the femoral head and the acetabulum, appears promising for the relief of pain without sacrificing hip joint function. This is an ingenious procedure especially applicable to the treatment of advanced hypertrophic arthritis of the hip. It is a relatively new procedure, however, and its exact place in the therapy of hip joint disease and the end results are still to be evaluated precisely.

USEFUL MEASURES IN THE TREATMENT OF HYPERTROPHIC ARTHRITIS OF THE SPINE

Relief of the symptoms induced by hypertrophic arthritis of the cervical spine, especially the headache so frequently associated with it, requires both systemic and local treatment. Fatigue must be eliminated. All systemic factors that might be contributory require attention, particularly existing anemia, hypothyroidism, focal sepsis. Such patients should avoid exposure to excessive cold and protect themselves against drafts. Ocular defects should be corrected to eliminate eye strain. Simple heat over the cervical and occipital regions, followed by massage, may be amazingly successful. Physiotherapy is particularly helpful if it is applied frequently, at least once or twice a day. Deep massage has been suggested to break up the fibrositic nodules frequently associated. Salicylates combined with heat, employed shortly after the onset of headache in the morning, sometimes afford prompt relief. Some patients state that 5 or 10 grains of acetylsalicylic acid, taken before retiring, may avert headache the following morning. A low carbohydrate diet, the use of vitamin B, and the use of saline laxatives for maintenance of regular action of the bowels, have been recommended, but it is difficult to be sure just what their place is in the therapeutic regimen.

When climatic conditions appear to be definitely related etiologically

once a week for from four to eight weeks, followed by smaller doses by mouth for a more extended period. The injection of massive doses of estrogenic hormone over long periods of time may not be devoid of certain undesirable effects. Such dosage has been suspected of producing ovarian cysts, cystic mastitis and perhaps even carcinomatous change in the breast.

PREVENTION AND TREATMENT OF DEFORMITIES

The principles that govern the prevention and treatment of deformities in chronic arthritis have already been discussed (page 167). The same principles that apply to the care of the joints in atrophic arthritis apply also to those in hypertrophic (osteo) arthritis. There are, however, variations in detail dictated by the differences in the pathologic process in each of these types of rheumatic disease.

We have emphasized that the pathologic changes in hypertrophic arthritis are of a bland noninflammatory type. Hyperplasia of the synovial membrane and its villi may develop, but it is quite different from the inflammatory pannus of atrophic arthritis. Cartilage degeneration occurs, but the hypertrophic bony changes are the more prominent end result. Unlike the situation in atrophic arthritis, muscle spasm is a negligible factor, not seriously conducive to producing deformity. Muscle atrophy does not occur, except to that slight degree which inactivity forced by pain may induce. Hence the prevention of deformity in hypertrophic arthritis is really a simple matter. The natural tendency is for it not to occur at all.

Surgical Measures

When deformity and limitation of mobility do occur, they are largely the result of mechanical interference with joint function either from hypertrophic spurs, loose cartilaginous bodies, excessively thick, hyperplastic synovia or pain.

Surgical measures are necessary in those advanced cases in which extensive and permanent pathologic change has introduced deformity or interference with the usefulness of a joint. Synovectomy may be very helpful by restoring the normal range of mobility and by eliminating pain in cases of villous synovitis in which extreme synovial hypertrophy causes mechanical interference with smooth and normal function and in which resolution cannot be effected by rest and physiotherapy. Arthrotomy, for removal of loose cartilaginous bodies or for excision of large exostoses, with or without removal of the patella, may be necessary in those cases in which repeated locking of the joint occurs or in which extensive hypertrophic bony change interferes with the desired degree of flexion or extension.

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When climatic conditions appear to be definitely related etiologically

particularly if the headaches are incapacitating and occur only during the fall and winter being completely absent during the warm, summer months a change of climate is worth a trial

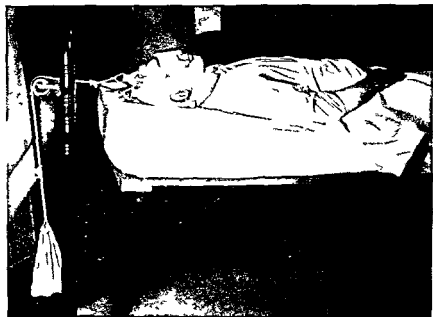


FIG 100 Continuous head traction applied in a case of hypertrophic arthritis of the cervical spine The head of the bed is elevated

When muscle spasm exists in association with hypertrophic arthritis at the cervical spine, relief may follow the use of a cervical (Thomas) collar (Fig 33) or continuous head traction with a Sayres sling (Fig 100) combined with physiotherapy, or stretching and manipulation of the cervical spine as advocated by Hanflig

In the latter procedure a block and tackle Sayres sling suspension apparatus is attached to an overhead hook With the patient seated on a chair under the apparatus the Sayres sling is applied over the head (Fig 101) Traction is then exerted until the patient's buttocks are lifted from the seat of the chair While the patient is thus suspended in the air, the head is rotated to one side then the other This procedure may be repeated two or three times in quick succession The patient is then lowered for a minute or two and the traction and manipulation are carried out once more

This treatment may be repeated at daily intervals or after two or three days depending upon the degree of reaction induced by the manipulation Physical therapy, including the application of radiant heat, hot compresses

or short wave diathermy and massage, are employed daily. For patients who are hospitalized, we prefer to employ continuous head traction during intervals between stretchings. When continuous traction is not employed a



FIG. 101 The apparatus, with the Sayres sling over the patient's head just before traction is applied for stretching and manipulation of the cervical spine

Thomas collar is applied. The patient wears this collar continuously, except during periods when physiotherapy is employed.

When pain results from an acute exacerbation of the arthritic process and there is marked muscle spasm and nuchal tenderness we believe forcible stretching and manipulation to be contraindicated. In such cases rest is enforced by splinting the spine either with a cervical collar or by immobilization in bed with continuous head traction.

Röntgenotherapy (page 242) has been suggested as another useful measure in such cases.

Paravertebral injection with procaine solution or in intractable cases, with alcohol may be required in older individuals who have severe pain associated with advanced hypertrophic arthritic change.

Surgical division of branches of the cervical plexus may be performed when there is intractable pain especially unilaterally, if it cannot be relieved by any other means.

In hypertrophic arthritis of the dorsal spine immobilization by a plaster jacket or Taylor brace may be required in addition to systemic measures and physiotherapy. Postural correction should be attempted particularly in younger patients.

Local measures applicable to the treatment of the low back or sciatic pain attributable to hypertrophic arthritis of the lumbar spine will be discussed more fully in a subsequent section (page 458).

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527.]

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PART IV

RHEUMATIC FEVER

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CHAPTER XXI

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SYNONYMS *Acute rheumatic fever inflammatory rheumatism the rheumatic state juvenile rheumatism*

Rheumatic fever is one of the most common types of acute rheumatic disease. Although the specific etiologic factor is unknown, rheumatic fever appears to be an infectious process; the most characteristic features of which, especially during acute stages, are fever and inflammatory changes in joints, frequently followed by carditis. The condition may develop at any age, but occurs most frequently in children and young adults; approximately 90 per cent of all cases develop between the ages of five and fifteen years. As in atrophic arthritis, there is a somewhat greater susceptibility to the disease among females than males.

From the standpoint of the damage to joint structures, rheumatic fever is not serious, but because of its tendency to involve the heart, it ranks among the most serious and fatal diseases of childhood and adolescence.

ETIOLOGY

Through the years there has accumulated increasing bacteriologic and immunologic evidence which, though not conclusive, indicates the probability that the hemolytic streptococcus may eventually be established as the precipitating etiologic factor in rheumatic fever as well as in atrophic arthritis.

Evidence has also been presented suggesting that a virus may possibly be related etiologically. These data are again by no means conclusive and represent merely another clue that deserves further investigation. The possibility exists that if a virus is responsible, it may be in some manner related to the streptococcus. Studies on the possible virus etiology of rheumatic disease have been dealt with in a previous section (page 37) and among the most comprehensive reviews on the bacteriologic and immunologic aspects of the etiology of rheumatic fever is that of Swift (1936).

Although the microbic origin of rheumatic fever receives support, it must not be assumed that infection alone is the beginning and the end of the

etiology of this disease. Again, as in atrophic arthritis, factors of inherent susceptibility, nutritional disturbances, vitamin deficiency states, debility from any cause and many other extraneous influences appear to play a role. Although data on these factors are not as complete as they might be, there are indications that they are as important in the development of rheumatic fever as of atrophic arthritis.

FAMILIAL SUSCEPTIBILITY

There is a striking familial tendency to the occurrence of rheumatic fever, the disease developing much more frequently in families of rheumatic children than in others. In half of the cases observed, two or more members of the family are affected by this malady, whereas only one in ten cases occurs in families in which other members do not have rheumatism. This family tendency is also reflected in the greater incidence of rheumatic heart disease among those living in contact with rheumatic infection as compared with the incidence of rheumatic heart disease in the general population.

Just what is responsible for the high familial tendency to rheumatic fever is not entirely clear. It may be the result of an inherent susceptibility to it, or the rheumatic infection may perhaps be transmitted by contagion from one member to another, or it may be that given a certain predisposition several members of the same family are affected because they are equally exposed to the same unfavorable hygienic, climatic, and other environmental factors.

NUTRITIONAL DISTURBANCES AND VITAMIN DEFICIENCY

As predisposing factors in rheumatic fever nutritional disturbances appear to be significant, but just how they increase susceptibility is not specifically known. There is accumulating evidence that vitamin deficiency, especially that of vitamin C, may be in some measure responsible for the occurrence of rheumatic fever, as has been suggested by the work of Rinehart and his co-workers, and others. Such vitamin deficiency may be the cause or merely the result of the rheumatic infection. However, the clinical association of the two is obvious and probably represents more than a chance association.

Nutritional disturbances, likely to be more prevalent among the poor, may explain in some measure the greater incidence of rheumatic fever among those in the lower economic strata. There are other factors at work, however, among such persons, which can conceivably influence their greater susceptibility to rheumatic infection. Unfavorable hygienic conditions, ex-

posure to cold and damp, and poor housing and overcrowding leading to increased exposure to spread of respiratory infections

PATHOLOGY AND PATHOGENESIS

The pathologic changes in rheumatic fever are by no means confined to the joints and heart, but involve many tissues and organs in the body. Although the most obvious lesions usually develop predominantly in articular structures or the heart, there are instances in which the pathologic change is most pronounced elsewhere. For example, the rheumatic process may be manifest predominantly in the abdominal viscera and peritoneum when other rheumatic lesions are hardly perceptible clinically.

The essential pathologic change is one of damage to mesenchymal ground substance of the affected tissue. *Focal edema, fibrinoid swelling and necrosis* occur first in the interstitial connective tissue, a change which apparently provokes the subsequent exudative and proliferative cellular reactions. Whether one or the other predominates, depends apparently upon the capacity of the tissue for one type of response or another. Thus, the proliferative type of reaction is especially prominent in the myocardium, endocardium, lungs, and subcutaneous tissues. The exudative reaction on the other hand, is to be noted especially in joint structures, pericardium, and pleura, leading to joint effusion, pericarditis or pleurisy. From a practical standpoint it is significant that the eventual tissue damage is apt to be greater where a proliferative, rather than an exudative, reaction predominates. The tendency to complete restoration to normal is therefore greater in joints subjected to rheumatic joint inflammation associated with effusion than in heart muscle or endocardium which had been the seat of a proliferative type of tissue reaction. The latter results in the formation of the characteristic Aschoff nodules, collections of large, round and ovoid wandering cells which have the tendency to group themselves concentrically around the smaller blood vessels. The characteristic morphologic appearance of the Aschoff nodule makes it the most easily recognized pathologic manifestation of rheumatic disease.

The dense scarring observed in the heart valves, myocardium, lungs and elsewhere represents the final phase in the pathologic process, resulting from fibrosis of previously active proliferative foci.

The clinical relationship between upper respiratory infections (especially tonsillitis) and the onset and recrudescence of acute episodes of rheumatic infection is pretty well established. It is not clear whether the disease process results from direct invasion of distant tissues by organisms arising from the primary focus of infection, or whether the pathologic change represents more of an allergic (hyperergic) response to the organism or its toxic prod-

etiology of this disease. Again as in atrophic arthritis, factors of inherent susceptibility, nutritional disturbances, vitamin deficiency states, debility from any cause and many other extraneous influences appear to play a role. Although data on these factors are not as complete as they might be, there are indications that they are as important in the development of rheumatic fever as of atrophic arthritis.

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uct As to the relationship between various foci of infection and rheumatic fever there are divergent opinions We have already discussed the pros and cons concerning the relation of focal infection to atrophic arthritis (pages 65-74) considerations which apply equally to the problem of rheumatic fever

III. RELATIONSHIP BETWEEN RHEUMATIC FEVER AND ATROPHIC ARTHRITIS

Although the typical clinical picture of rheumatic fever is quite different from that of atrophic arthritis, many facts suggest that pathologically, and perhaps etiologically also these two diseases may not be unrelated This suspicion is strengthened especially by borderline cases, encountered not infrequently in which the clinical and pathologic manifestations present essentially a composite of certain manifestations of rheumatic fever and atrophic arthritis The possible relationship becomes especially evident in transitional cases in which a typical acute rheumatic process is transformed into a chronic form of atrophic arthritis with the eventual development of typical cardiac lesions of rheumatism as well as the characteristic pathologic changes and deformities of atrophic arthritis

From data available at present it appears not illogical to picture acute rheumatic fever and atrophic arthritis as facets of an essentially unitary pathologic process the differences in the clinical and pathologic reactions of which may be conditioned perhaps by variations in inherent susceptibility the age of the patient, and the environmental factors under which the infection occurred In this connection it is important to recall that bacteriologic and immunologic studies as well as evidence relating to the pathogenesis in these two diseases have shown for the most part a remarkable degree of parallelism if not actual similarity True, certain immunologic reactions in atrophic arthritis differ from those in rheumatic fever In the first for example there is usually a low antistreptolysin content in the serum and a high agglutinin titer for streptococci whereas in rheumatic fever the contrary is true This difference may perhaps be attributable to the acuteness of the process in rheumatic fever and its relative chronicity in atrophic arthritis For in early, active cases of the latter the antistreptolysin content of the serum is sometimes appreciable, even though it may not attain the proportion noted in acute rheumatic fever Also, the remarkable similarity in the clinical and pathologic features of the subcutaneous nodules in rheumatic fever and atrophic arthritis, demonstrated by Dawson furnishes additional proof that at least with regard to this phase of the pathologic process the two diseases are basically similar, even though there may be differences in degree of reaction

The possibility that both rheumatic fever and atrophic arthritis may represent variations of the same pathologic process suggests that studies in one disease may be helpful in elucidating the nature of the other. Nevertheless such a conception does not alter the basic fact that these two diseases present distinct problems from the standpoint of diagnosis, treatment and prognosis.

CLINICAL MANIFESTATIONS

The mode of onset and the course of rheumatic fever vary greatly in different patients depending upon the severity and acuteness of the process which are in turn conditioned by the age of the individual affected.

In over half the cases the onset of the disease is preceded by an acute respiratory infection often by an attack of sore throat or tonsillitis. The first manifestations of the rheumatic process may appear within a few days after the respiratory infection or not for a week or two afterward. If the onset is very abrupt the patient may suddenly develop a chill, fever ranging between 100° and 104° F., a rapid pulse, pallor, profuse sweating, evidence of toxemia, sometimes with severe prostration and pain in various joints.

There is a tendency for several of the larger joints to become involved simultaneously or in close succession and for a migratory type of involvement of new joints as previously affected ones clear. In most cases there is only periarticular swelling but in exceptional instances effusion of fluid into the joint does occur. In still others the pain and tenderness are accompanied by swelling so slight in degree as to be hardly perceptible clinically. The joint pain is generally severe and the tenderness exquisite so that the slightest pressure even of the bedclothes may be intolerable. Redness over the joints and perceptible local heat are apparent in only the most acute stages.

The onset of the disease may be more gradual, beginning with only mild tenderness and aching about a joint leading to an erroneous suspicion of a sprain. Such symptoms may persist for several days or a week at which time several joints may become acutely inflamed and evidence of an acute systemic infection is apparent.

In young children the articular pain may be relatively mild and evanescent and not confined strictly to the joints whereas in older individuals the arthritis is apt to present a more striking part of the clinical picture.

In children in whom the onset of the disease occurs gradually and not explosively there may be no localizable joint symptoms at all, only a vague arthralgia, perhaps muscular pain, epistaxis, abdominal discomfort, evidence of general malaise and the development of pronounced pallor followed by the appearance of a low grade fever, tachycardia out of proportion to the

rise in temperature, then night sweats, subcutaneous nodules and cardiac murmurs

Unless one is particularly alert to the possibility of such types of rheumatic manifestation in the young, the active stage of the process may pass undetected for a long time, leaving the way open for the development of cardiac damage, which might perhaps have been averted in some of them. Very likely this atypical form of rheumatic infection, undetected during its active stages, accounts for those many cases of rheumatic valvulitis discovered in later life in which a clear cut history of rheumatic fever cannot be obtained.

The disease may run a relatively short course, the joint pains disappearing within five to ten days after the institution of treatment with salicylates. Defervescence may be delayed for several days longer, then all the other residual symptoms may disappear, and the attack subside.

Again the initial attack may become protracted, the joint swelling persisting despite intensive treatment and suggesting the possibility of atrophic arthritis. Eventually all of the symptoms (including the arthritis, fever, and toxemia) may abate only to flare again into renewed activity, sometimes with the development of increased toxemia, more acceleration of the pulse, augmentation of dyspnea and development of precordial pain indicating the onset or exacerbation of cardiac involvement. Such symptoms may persist again for days or weeks, until the activity of the rheumatic process abates again. Or the severity of the infection may continue to increase, the cardiac damage becoming progressively more pronounced and alarming, with fatal results.

The so-called 'growing pains' occurring in children of poor general health and presenting some of the stigmata of rheumatic infection, such as nose bleeds, slight fever, pallor and undernutrition, may actually be manifestations of subacute rheumatic fever. Such cases should be differentiated from others with pains in the extremities, which apparently may occur in growing children without the slightest evidence of rheumatic infection. Shapiro states that nonrheumatic pains in children occur most commonly in the muscles of the legs and develop either at the end of the day or at night, sometimes awakening the child during sleep, but such pain is usually gone by morning, and does not recur through the day, there are no other signs of rheumatism elsewhere, nor a family history of rheumatic fever. The benign character of such pains is established further by a normal sedimentation rate and blood count. The so-called "growing pains" representing actually manifestations of subacute rheumatic fever are apt to appear early in the morning, to persist all day, to be aggravated by activity, and to be relieved by rest and heat. Moreover, such rheumatic pains appear in the upper as well as the lower extremities, there are other signs of rheumatic

infection such as an increased rate of sedimentation leucocytosis anemia and very likely a family history of rheumatism

The tendency of rheumatic fever to recur is well known. When recurrences follow one another in close succession it may be that the repeated flare ups represent merely reactivation in patients in whom the previous episode was not entirely burned out. In others recurrences develop months or years later induced by upper respiratory infection as a rule though appearing not infrequently without any apparent reason. Recurrent bouts of rheumatic infection are more likely in younger than older patients. These recurrent infections increase the tendency to cardiac damage aggravating cardiac defects that occurred during the first attack or initiating carditis if the patient had previously escaped it.

Skin Manifestations

Subcutaneous nodules and a variety of other cutaneous manifestations develop in a small proportion of cases of rheumatic fever. The occurrence of erythema nodosum erythema multiforme and other types of skin eruptions apparently represent reactions of the skin to the toxemia of the systemic infection and indicate as a rule relatively virulent forms of rheumatism.

Abdominal Manifestations

Although mild abdominal pains or epigastric cramps sometimes associated with nausea and vomiting and slight fever occur frequently as manifestations of rheumatic infection severe abdominal pain simulating attacks of acute appendicitis or any other acute abdominal disease may occur as the most prominent or sole manifestation. The severity of the abdominal pain as well as the pattern of the acute attack may divert attention from a number of vague prodromal symptoms suggestive of rheumatic infection. The patient may be operated upon for appendicitis the diagnosis of rheumatic abdominal infection becoming evident at operation or unfortunately during subsequent autopsy. In some cases the manifestations resemble acute appendicitis so closely that operation is justified and should be performed even when the suspicion of a possible abdominal rheumatic infection is considered but cannot be proved without exploration. Worthy of note is the fact that the erythrocyte sedimentation rate is increased in rheumatic fever while it is normal in uncomplicated acute appendicitis. If the acute episode of abdominal rheumatism subsides spontaneously it may recur.

In a case of abdominal rheumatic fever seen by the writer recently the acute abdominal symptoms were extremely suggestive of appendicitis. Fortunately operation had been deferred by the surgeon because when he

first saw the patient three days after the onset of the attack there were generalized abdominal rigidity tenderness and distention suggesting the possibility of peritonitis presumed to have resulted from a perforated appendix. On the seventh day however severe pain and swelling developed in several joints arousing the suspicion that the entire process was on a rheumatic basis. Treatment with salicylates administered per rectum promptly resulted in a drop of temperature amelioration of abdominal symptoms and signs and complete recovery during the following week. Review of the previous medical history brought out the occurrence of similar attacks of abdominal pain on two previous occasions and other suggestive signs of a low grade rheumatic process.

Rheumatic Pneumonia

Bronchopneumonic lesions sometimes extensive consolidation followed by massive collapse of the lungs may develop in the course of rheumatic infection. Rheumatic pleurisy is not an uncommon complication. The pneumonic lesions of rheumatic fever may be difficult to detect clinically because they have a tendency to be evanescent and the physical findings atypical and transitory.

Cardiac Involvement

Precordial pain dyspnea marked acceleration of the pulse a pericardial friction rub the development of cardiac murmurs or enlargement of the heart—any of these signs indicate the presence of cardiac involvement. Cardiac murmurs may of course be induced by a temporary toxic myocarditis but occurring in the course of active rheumatic fever they must be regarded as a possible sign of more serious damage. Rheumatic endocarditis affects the mitral valve most commonly the aortic valve next often and not infrequently both. Although evidence of valvulitis may predominate a pancarditis usually exists. In some cases the brunt of the damage is inflicted upon the pericardium or myocardium eventually resulting in crippling adhesive pericarditis or cardiac insufficiency and arrhythmias. There may be no distinctive clinical signs of cardiac involvement when electrocardiographic evidence of delayed or disturbed conduction may indicate implication of cardiac muscle and its conduction apparatus.

In other cases the heart escapes involvement altogether during the first attack, only to succumb during a subsequent one. Naturally the greater the number of recurrences the greater the likelihood of cardiac involvement. Also the younger the patient the more likely is the heart to be affected.

The attack of rheumatic fever may cause such slight damage to the heart valves or muscle as to be completely healed and imperceptible to clinical examination or a somewhat greater degree of valvular damage may result

with stenosis of the valve supervening five to seven years later. Yet if the damage to the myocardium has not been too severe the patient may live through his normal life expectancy without serious cardiac embarrassment. This is especially likely to occur if there is knowledge of the existence of the cardiac lesion from its inception and if adequate protection is provided to maintain a normal cardiac reserve and the prevention of recurrent attacks of rheumatic fever.

Finally rheumatic fever may jeopardize the patient's life indirectly through the development of subacute or malignant bacterial endocarditis engrafted upon a previously damaged (though relatively benign) rheumatic valvulitis.

LABORATORY AIDS IN DIAGNOSIS

During the acute stage of rheumatic fever the blood count generally reveals a leucocytosis, the count ranging from 10 000 to 20 000 cells; it may be higher if the infection is of a severe grade and associated with high fever. The proportion of polymorphonuclear leucocytes is moderately increased. The leucocytosis persists through the period of active infection, the blood count returning to normal with subsidence of the process. A hypochromic type of anemia is not uncommon, especially in children.

The rate of sedimentation of erythrocytes is invariably accelerated during active stages of the process, the rate slowing to normal as activity subsides. The technique for the performance of this test and the interpretation of the results have already been described (page 97).

Cultures of joint fluid or blood by ordinary methods do not yield any growth of organisms.

Serologic studies generally reveal high antistreptolysin and antifibrinolysin titers but relatively low agglutinin titers. Such serologic tests requiring involved technical procedures do not constitute practical aids in the diagnosis of rheumatic fever.

The formol gel reaction first described by Gate and Papacostas is a simple laboratory procedure which appears to offer valuable aid in the detection of active rheumatic carditis. The test is based essentially on the principle that in rheumatic fever, especially when there is active carditis and hyperglobulinemia, the addition of formalin to a sample of the blood serum results in gelation of the serum, with or without the development of opacity. Significantly, the formol gel reaction may be negative during the early stages of the disease, even though the erythrocyte sedimentation rate is accelerated; with the development of active carditis a formol gel reaction appears, remaining positive so long as there is active carditis, even though the sedimentation rate may return to normal.

Technique for Performance of the Formol gel Test

Schultz and Rose describe the technique for this test as follows

In performing the test two drops of 40 per cent formalin were added, with shaking to a test tube of 8 mm bore containing 1.0 cc of the serum to be examined. The tube was allowed to stand at room temperature and the contents were inspected for gelation and opacity at 5 minutes, 2 hours and 24 hours. Strongly positive sera develop alterations in physical state at 2 hours and occasionally at 5 minutes but the results reported are exclusively those of the 24 hour reading. The criteria suggested by Gutman and Wise were observed in estimating the intensity (+ to + + + +) of gelation or opacity. Serum was obtained by allowing venous blood, aseptically drawn from the antecubital region with a minimum of stasis to clot in paraffin lined tubes at room temperature. In almost all instances specimens were collected before breakfast in order to obtain clear serum.

In a study of the formol gel reaction in seventy patients with rheumatic fever Schultz and Rose concluded that strongly positive reactions in rheumatic children or in adults without arthritis suggest the presence of active carditis whereas persistently negative results indicate the absence of severe carditis. They feel that a positive formol gel reaction, persisting after the sedimentation rate has returned to normal may provide the only evidence of continued rheumatic activity indicating the necessity for continued rest to prevent cardiac damage.

ROENTGENOGRAPHIC FINDINGS

Roentgenographic study in rheumatic fever adds nothing of diagnostic significance. There are of course no changes in the cartilage and bone. If periarthritic swelling or effusion of fluid into the joint has developed evidence of these may be present but cannot be employed for differentiating between rheumatic fever and early atrophic arthritis.

ELECTROCARDIOGRAPHIC STUDY

Abnormal electrocardiographic findings, observed so often in rheumatic fever may constitute important evidence favoring a diagnosis of rheumatic fever when the possibility of atrophic arthritis is to be excluded. In the latter, significant electrocardiographic abnormalities are unusual, in rheumatic fever, on the other hand the electrocardiogram frequently reveals some form of abnormality. Cohn and Swift (1924) and Master and Jaffe (1932) have shown that electrocardiographic tracings obtained at frequent

intervals during the course of rheumatic fever reveal abnormal changes, at some time or other, in practically all cases. Increased auriculoventricular conduction time, various grades of intraventricular block, abnormalities in the R S T and Q R S complexes, inversions of the T wave, auricular fibrillation or flutter—any such abnormalities, and others, may be encountered.

DIFFERENTIAL DIAGNOSIS

Although the history and the clinical examination usually suffice for establishing the diagnosis of rheumatic fever, cases occur in which the exclusion of other types of acute arthritis may be necessary.

The differentiation between some cases of rheumatic fever and atrophic arthritis is not always a simple matter. In some of these, the differential diagnosis may be impossible, except after a long period of observation, during which the clinical picture may at one time suggest rheumatic fever and, at another, atrophic arthritis. Eventually, however, the diagnosis becomes clear: either frank endocarditis develops, or the condition progresses to a state of chronicity, with permanent changes in the joints. Other features of the differential diagnosis between these two conditions have been discussed previously (page 107).

The occurrence of repeated bouts of acute arthritis in an adult, with complete disappearance of joint manifestations after each attack, may suggest the diagnosis of gout. Differentiation should not be difficult, however, for in gout there is likely to be involvement of the great toe joint, the possible presence of tophi, characteristic roentgenographic changes, as well as an increased uric acid concentration in the blood. After several recurrences of rheumatic fever there is apt to be evidence of valvular damage, whereas in gout there is likely to be evidence of nephritis.

An erroneous diagnosis of rheumatic fever may be made if gonococcal arthritis is preceded by an acute upper respiratory infection. Many clinical features of gonococcal arthritis should serve, however, to distinguish it from rheumatic fever and aid may be obtained from specific laboratory studies (see page 328).

The polyarthritis of serum sickness may resemble rheumatic fever very closely, but the history of a recent injection of serum and the associated signs of serum sickness serve to differentiate the two.

TREATMENT

The treatment of rheumatic fever must aim not only to relieve the patient of the pain of the arthritis, but must be directed toward inactivating the

disease completely so that carditis may be prevented. The patient must be kept strictly in bed until every trace of activity of the rheumatic process has disappeared—that is until the patient is not only free of clinical manifestations of his disease but also presents a normal temperature, white blood count and sedimentation rate. As we have already indicated the possibility of residual active carditis exists even though the sedimentation rate has become normal. In such cases disappearance of the formol gel reaction is to be desired in addition.

It is worth enforcing rest no matter how long it may be required to inactivate the disease completely for nothing can take the place of rest for the prevention of cardiac involvement and for healing any lesions that may have developed. When completely normal conditions have existed for ten days or two weeks the patient may be allowed out of bed the temperature and pulse rate being observed. If a rise in temperature, acceleration of the pulse or increase in the rate of sedimentation occurs the patient must return to bed until a normal status is again attained.

During convalescence care must be taken to prevent strain on the heart especially if there had been signs of carditis no matter how trivial. The experience with convalescent homes established in some parts of the United States and in England has shown their value in the prevention of recurrent rheumatism and in reducing the severity of cardiac crippling.

Local therapy. The affected joints should be placed in a position of maximum relaxation the position of slight flexion being sometimes necessary to secure comfort. The limbs may be maintained in this position by pillows splints being generally unnecessary. During the acute stage the joints should of course be moved as little as possible protection even from the weight of bedclothes should be afforded. To decrease pain the affected joints may be wrapped in cotton or wool after the application of a 10 per cent ointment of methyl salicylate or hot compresses of saturated solution of magnesium sulphate may be applied.

Diet. During the early stage of the illness a soft diet is allowed. Later a general diet is prescribed. In view of Rinchart's observations on the possible relationship of vitamin C deficiency to the rheumatic state it seems logical to administer liberal amounts of citrus fruits and fruit juices and even supplementary doses of 0.1 to 0.5 gm. daily of ascorbic (cevitamic) acid.

Medicinal treatment. The salicylic acid compounds occupy an important place in the medicinal treatment of rheumatic fever. Although the mode of action of salicylates is unknown they act practically as specifics for the prompt alleviation of pain swelling in the joints and reduction of fever.

In rheumatic fever relief follows the administration of salicylates so

consistently that failure to obtain satisfactory therapeutic results should arouse the suspicion that one is dealing with a form of arthritis other than rheumatic fever

Sodium salicylate is most commonly employed, being administered every two to three hours in doses of 15 to 20 grains, combined with an equal amount of sodium bicarbonate. Such administration is continued until the symptoms are controlled, unless nausea, vomiting, disturbances of vision, ringing in the ears, delirium, or albuminuria appear, in which case the drug is discontinued.

In very acute cases 15 to 20 grains of sodium salicylate may be administered every hour for 8 or 10 doses, unless symptoms of drug intolerance appear.

Acetylsalicylic acid in doses of 15 to 20 grains, every two hours, may be employed instead of sodium salicylate, and with equally satisfactory results. Sometimes acetylsalicylic acid is better tolerated than sodium salicylate.

Aminopyrine in doses of 5 to 10 grains every four to six hours has also been employed. Some patients have an idiosyncrasy to this drug, however, developing granulocytopenia or agranulocytosis. Therefore, the blood count should be checked at frequent intervals, if aminopyrine is employed.

If the pain is very severe, as it may be at the onset, the administration of codeine sulphate or morphine may be required.

Swift and his co-workers (1938) have shown that sulfanilamide has no place in the treatment of rheumatic fever. Coburn and Moore found, however, that maintenance doses (about 30 grains daily) of sulfanilamide, administered over a period of months greatly lessened the incidence of hemolytic streptococcus infection and recurrences of rheumatic fever. They emphasized that sulfanilamide administered after the onset of streptococcal throat infections did not prevent recurrences of the rheumatic disease.

Even when evidence of cardiac involvement exists, the use of *digitalis* is not required, unless signs of cardiac failure or fibrillation appear.

Since anemia is a frequent concomitant of rheumatic infection, the administration of some form of iron preparation, in adequate dosage, is generally necessary during convalescence. *Ferrous sulphate* in doses of 3 grains, four times a day, or *ferric* and *ammonium citrate* in doses of $7\frac{1}{2}$ to 15 grains, three or four times a day, may be employed. Small blood transfusions may be necessary if the anemia is severe. Solution of potassium arsenite in doses of 1 minim, increased to 5 minims, three times a day, may be employed in addition to the iron.

Treatment of focal infection. Although difference of opinion exists concerning the relation of focal infection to rheumatic fever, and especially concerning the value of eradicating focal infection after the disease has become established, the consensus of opinion is that focal infection, wherever it

may exist should be eradicated promptly after subsidence of the acute process. The removal of diseased tonsils is especially to be recommended. Tonsillectomy would seem justifiable in most cases of rheumatic fever even when there is merely a suspicion that the tonsils are infected. We recognize that neither tonsillectomy nor the eradication of other foci of infection insures against recurrent episodes of rheumatic fever. We feel that focal infection is as important in relation to rheumatic fever as to atrophic arthritis. This phase of the subject has been covered in considerable detail in previous sections (pages 65 and 74).

Vaccines and other types of antigenic therapy. Intravenous injection of *streptococcus vaccine filtrates* and *antistreptococcus serum* has been recommended but the results of such therapy are too inconclusive to warrant their employment in general practice. *Nonspecific protein therapy* is not recommended.

Climatotherapy. Since the incidence of rheumatic fever is to a large extent determined by the occurrence of upper respiratory infections it is understandable why rheumatic patients transported to tropical or subtropical climates where they are more free of such infections are less subject to recurrence of rheumatic disease. Although not absolutely insured against persistence of active rheumatic infection or its recurrence such a change of climate appears to lessen the severity of the disease and the tendency to recurrence but only so long as the patient remains in the tropics. Obviously then climatotherapy does not provide a practical solution to the general problem of rheumatic disease.

Physical therapy. Physical therapy is not called for during the acute stages of the disease but may be employed in chronic forms. Fever therapy has been employed advantageously in the treatment of rheumatic fever and chorea but its place is limited.

Preventive measures.—Prophylactic measures against rheumatic fever should occupy a much larger place in the therapeutic scheme than they do now. Although important in all types of rheumatism prophylaxis is especially to be applied in rheumatic fever in order that recurrent attacks with each of which goes an increased likelihood of cardiac damage may be averted.

The general hygienic measures to be employed applicable also to the prevention of rheumatic disease in general have already been discussed (page 155).

The provision of adequate and sanitary housing, warm clothing, sunlight and optimum nutrition is basically important. Susceptible subjects should avoid overcrowding, exposure to colds and close contact with those suffering from upper respiratory infections. The early eradication of focal infection especially tonsillar sepsis seems of practical value. Children with

a rheumatic and allergic diathesis (which are frequently associated) should have existing allergic conditions in the upper respiratory tract corrected, so as to lessen the vulnerability to superimposed infection

The value of prophylactic vaccination against upper respiratory infections and rheumatism has not been established

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[For a list of books and monographs dealing with the general aspects of

chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527]

A Virus in the Etiology

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PART V

SPECIFIC ARTHRITIDES

GOUTY ARTHRITIS

GONOCOCCAL ARTHRITIS

TUBERCULOUS ARTHRITIS

SOME LESS COMMON FORMS OF SPECIFIC INFECTIOUS ARTHRITIS

Acute Suppurative Arthritis

Pneumococcal Arthritis

The Arthritis of Meningococcus (Cerebrospinal) Meningitis

Arthritis of Scarlet Fever (Scarlatinal Rheumatism)

Syphilitic Arthritis

The Arthritis of Brucellosis

The Arthritis of Haverhill Fever (Erythema Arthriticum Epidemicum)

Arthritis Associated with Lymphogranuloma Venereum

Arthritis Associated with Ulcerative Colitis

Arthritis of Typhoid Fever

Tuberculous Rheumatism

Other Forms of Specific Infectious Arthritis

NOTES ON SOME FORMS OF NONINFECTIOUS SPECIFIC ARTHRITIS

Traumatic Arthritis

Hemophilic Arthritis

Allergic Arthritis (Arthritis of Serum Sickness)

Neuropathic (Charcot) Joint Disease

CHAPTER XXII

GOUTY ARTHRITIS

Because of an increased interest in gout in recent years we have become aware that it is not at all uncommon. Those familiar with its clinical manifestations discover many cases formerly treated as nonspecific arthritis.

Gout is a disturbance in which the metabolism of uric acid and its precursors, the purines, is largely affected. The clinical manifestation most evident is arthritis, due to deposition of urates in and about the joints. In most instances there is no difficulty in diagnosing gout provided its possibility is considered. Since the treatment of gout differs so radically from that of all other forms of arthritis, and is so efficacious, accuracy in diagnosis is essential to therapeutic success.

In the diagnosis of this type of arthritis the patient's history is most important. One may go so far as to say that with the characteristic history of gout, the condition may often be diagnosed correctly even when some of the most pathognomonic signs of the disease are absent.

HEREDITY

The long recognized hereditary tendency to the gouty diathesis is frequently evident, though in many cases a positive family history of gout is not elicited. Among the latter, one may find, however, a familial tendency to other rheumatic disorders, migraine, or allergic diatheses. An interesting and probably significant observation in this connection is that of Jacobson who noted elevated serum uric acid levels in non-gouty relatives of gouty individuals. These findings suggest an inherent tendency to a disturbance in uric acid metabolism, which may remain temporarily or permanently latent. Such findings also suggest that in the absence of clinical gout, renal insufficiency, disease of the liver, or leucemia, any of which may produce elevated uric acid levels, the possibility of latent, asymptomatic gout, or at least an inherent tendency to it, must be thought of when hyperuricemia is found.

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SOME ASPECTS OF THE METABOLISM

Although it is clear that some disturbance of uric acid metabolism is primarily related to gout one does not always find a relation between the amount of uric acid in the blood and the occurrence or severity of attacks. It is possible although unusual for individuals with gout to have a persistently low content of uric acid in the blood and to have typical acute attacks without change in either the uric acid content or its excretion. This indicates that the content of uric acid in the blood does not always reflect what transpires in the joint tissues during acute exacerbations of the disease. This may be explained on the basis of a possible heightened tendency for the absorption of urates by the tissues even at times when the blood does not supply an excess of uric acid. Hence we cannot look upon gout as merely a result of retention of uric acid consequent to defective elimination of this substance through the kidneys. Patients with early gout show no impairment in excretion of uric acid. Impairment of renal function which frequently develops in the later stages of gout is evidently a result of kidney damage induced by excessive excretion of uric acid over long periods of time.

Falbott, Jacobson and Oberg and Falbott and Coombs have demonstrated cyclic variations in water and salt excretion in individuals with gout. Studying a group of patients who were on a constant intake of food and fluid Falbott noted that diuresis preceded acute attacks of gout by twenty-four to seventy-two hours. During the height of the diuresis which occurred the day before or coincident with the onset of articular symptoms the output of fluid was approximately double the daily output prior to the onset of the diuresis. There was also an increased output of urates, sodium chloride and other electrolytes. The periods of heightened water and salt excretion were followed by periods of diminished excretion and by cyclic recurrence of the diuresis phenomenon at subsequent times. These variations occurred not only in relation to acute attacks of gout but also during arthritis-free intervals. It is interesting that despite increased diuresis an increase in body weight occurred, which could be correlated with a proportionate diminution in insensible weight loss. These observations offer an explanation for the belief expressed by certain patients that they can predict recurrences of acute gout from an increased urinary output, suppression of sweating or a gain in body weight. Further observation revealed that such reduction of insensible weight loss could be correlated chronologically with falls in barometric pressure. Fluctuations in environmental temperature and humidity were not so related.

INCIDENCE

The fourth decade is the most common period for the onset of gouty arthritis. It may also occur in younger individuals, in which case the condition may be polyarticular, and even migratory in character, resembling rheumatic fever, it is likely to be severe with the attacks protracted and accompanied by fever. Such patients are especially susceptible to severe crippling; they are of course more liable to early visceral damage—arteriosclerosis and nephritis—which may shorten their lives.

Gout is relatively rare in women.

CLINICAL MANIFESTATIONS

Clinically, the course of gouty arthritis presents two phases: a relatively long period with acute recurrent attacks of the disease and finally a chronic stage, with permanent deformity of joints and other characteristic manifestations.

Attacks of gout may apparently be precipitated by physical trauma, by excesses in eating and drinking, by worry, or by prolonged application to nerve wracking mental tasks.

An acute attack of gout may be precipitated by an operation. Although the explanation for this relationship is not clear, the clinical association is frequent enough to warrant consideration of gout with any abrupt onset of arthritis shortly after a surgical operation.

The following report of a case of gout illustrates well the typical course and manifestations of the disease.

R. A. W. a man aged sixty, complained of rheumatism from which he had suffered for a number of years. The patient related that some eight years previously he had undergone an acute attack of arthritis of short duration which was confined to the right great toe. It had suddenly become extremely painful, sore and red, so that he was confined to bed for a week. During the subsequent two weeks the arthritis gradually subsided, and within three weeks from the time of onset, he was completely free of pain.

He then remained well until three years ago when, while in Florida, he had a recurrence of pain and inflammation in the previously affected great toe joint. Although the pain, swelling, redness and tenderness were quite pronounced, the attack again lasted a relatively short time, the entire process subsiding completely, leaving no residual changes in the joint. The subsequent winter he had a recurrence of similar trouble and again during the past winter, this last attack involving both great toe joints and the left elbow. There has developed recently a persistent thickening about the right great toe.

The essential features of these attacks, as the patient described them, were abrupt onset of joint pain, soreness, and extreme tenderness, relatively short duration of the attacks, complete subsidence of all evidence of arthritis at the conclusion of each attack, and total freedom from joint pains in the long intervals between attacks. Aside from the arthritic condition, the patient had no other symptoms except some dyspnea on moderate exertion.

The past history was negative except for pneumonia and typhoid fever when a young man, and a history of recurring attacks of abdominal pain, which he found were caused by certain foods, particularly liver and cabbage. Since refraining from these two food stuffs he has been practically free of indigestion. There was no history of an excessive use of alcoholic drinks. A number of years ago he was accustomed to drink wines in moderate quantities. In recent years he drank whiskey occasionally. He had never been a heavy beer drinker. There was no familial history of arthritis.

The physical examination revealed a patient of florid complexion with a slight tendency to obesity. He walked with a limp on account of the pain in the right great toe, and the right shoe was cut to relieve it from pressure. The pupils reacted normally. The teeth showed some evidence of infection. The tonsils appeared infected, liquid pus oozing when pressure was applied to the left tonsil. Over the left ear there were two small nodules. The larger, the size of a pea, appeared chalky white when the skin overlying it was stretched. Over the margin of the right ear there was a similar minute nodule.

All the joints appeared normal except the right toe, which was distinctly swollen, the result of thickening of perarticular structures. There were no signs of acute inflammation at this time and only a moderate degree of tenderness. There was some pain and limitation of motion at this joint. The left olecranon bursa appeared slightly thickened. The findings of the general examination were otherwise consistently negative.

Laboratory data. The routine blood count, and the Wassermann and Kahn tests on the serum were negative. Examination of the urine revealed a specific gravity of 1.010, a very faint trace of albumin, and an occasional leucocyte and coarsely granular cast in the sediment, but no erythrocytes. The blood sugar was 88 mg. and the blood uric acid was 6.6 mg. per 100 cc. of blood. The basal metabolic rate and gastric analysis did not reveal unusual findings.

Röntgenograms of the feet revealed punched-out areas in the metatarsophalangeal joints of both great toes, characteristic of gout. A roentgenogram of the left elbow was normal. The roentgenogram of the chest revealed slight dilatation of the ascending aorta, but the heart and lungs were normal. The dental examination was negative. An electrocardiogram revealed a prominent Q wave in lead III, and a poorly identified T wave in all leads, suggestive of coronary sclerosis. A minute amount of material removed from the tophus of the left ear when examined under the microscope, revealed the presence of the characteristic, needle-shaped urate crystals.

The final diagnosis was

1. Gout, entering the stage of chronicity.

- 2 Focal infection in teeth and tonsils
- 3 Mild chronic nephritis
- 4 Suggestion of coronary sclerosis

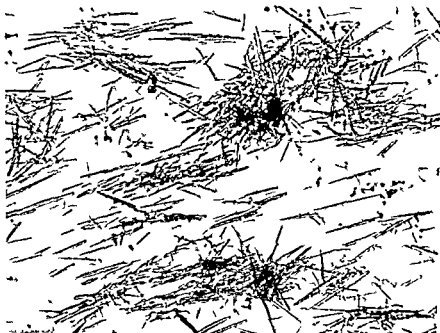


FIG 102 Typical needle shaped crystals of monosodium urate removed from a tophus in the ear of a gouty patient

This case presents many of the classical features of gouty arthritis. Not all cases are so typical, and for this reason we shall discuss briefly those features of the condition which appear even in the early stages of the disease, and those other manifestations of gout which may often be absent, even in fully developed cases.

As in the case just described, the characteristic history of gouty arthritis reveals recurrent bouts of acute or subacute arthritis, with severe pain, swelling and redness of joints, and exquisite tenderness. These attacks last a variable time, finally subsiding entirely, such paroxysms are separated by intervals of complete remission of joint activity. Especially significant is the absence of residua of arthritis during intermissions between paroxysms, before the appearance of chronic gout. This fact was emphasized long ago by Garrod and by Svedenham, who, incidentally, first segregated gouty arthritis from the maze of acute and chronic rheumatic diseases.

We have already mentioned the increased excretion of water, suppression of sweating and gain in weight, which may be noted preceding the onset of an acute attack of gout. Nausea, gastro-intestinal distress, nervous irrita-

bility, melancholia and vague muscular or joint pains and stiffness are also sometimes premonitory symptoms

Especially characteristic of the acute attack of gout is the abrupt develop-

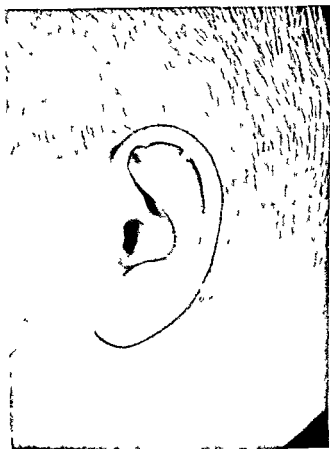


FIG. 103. Tophi in the helix of the ear of a patient with gouty arthritis. Material removed from one of these tophi revealed the typical monosodium urate crystals illustrated in Figure 102.

ment of joint swelling which may assume marked proportions within an hour or two of its onset. It may be so precipitate that the patient can actually refer to the specific hour when the attack began. The affected joints present the appearance of an acute inflammatory process suggesting the existence of septic arthritis or cellulitis. The area involved appears red, hot, tense, and glossy. The swelling generally extends for a distance beyond the joint. There is marked distention of the superficial veins, lymphangitis is occasionally an associated symptom. The pain is excruciating particularly at night. Because of the exquisite tenderness the patient fears the slightest touch, even of the bedclothes. The attack may last five to ten days and

though the more severe attacks in younger individuals may last for weeks. The earlier acute attacks are likely to be of shorter duration than the subsequent ones. With disappearance of the pain and swelling, local itching



FIG. 104. Large punched-out area in the head of the first metatarsal bone representing the typical roentgenographic picture of gouty arthritis.

sometimes develops. The patient may then remain entirely free of joint symptoms for months or years until the next attack.

One of the classical signs of gouty arthritis is the frequently observed tendency toward involvement of the great toe joints, a selective type of involvement particularly in initial attacks of the disease. It must be pointed

out, however, that here again exceptions to the rule are not uncommon. In approximately one third of some series of proved cases of gout, the initial attacks have occurred in joints other than the great toe. In any individual case, therefore, the situation of the arthritis is a much less dependable guide to diagnosis than are some of the other features of the disease.

Tophi, which constitute accumulations of urate crystals (Fig 102), are pathognomonic of gouty arthritis. Externally they are most often found on the cartilaginous part of the ears, usually at the helix (Fig 103). In advanced cases of gout the tophaceous material in the region of affected joints may form large masses, projecting under the skin, and even causing the overlying skin to ulcerate. While the finding of tophi in the ears or in other situations establishes the presence of gout, it must be remembered that in nearly 50 per cent of proved cases, tophi are entirely absent. They are not to be expected early in the disease. Even in cases of many years' duration and with marked characteristic changes in the joints, tophi may still not be evident. Therefore the presence of tophi is diagnostic, but their absence does not rule out the possibility of gout.

Punched-out areas in the cartilage or bones of the joint revealed in the roentgenogram are characteristic of gouty arthritis (Fig 104). If these erosions are small or if they occur in many joints of the feet or hands, particularly if they are symmetrically distributed, they should be interpreted with caution since they may also occur in rheumatoid arthritis. In the early stages of gout the roentgenogram usually reveals nothing abnormal, because the urates have not as yet been permanently deposited. When the typical roentgenographic evidence of gout appears, it is indicative of the advanced, chronic stage of the disease.

As a result of excessive excretion of urates by individuals with gout, the possibility of urate calculi formation is ever present. Renal colic is encountered in a small proportion of cases. But many more patients who develop urate calculi never develop clinical manifestations of gout.

DIAGNOSIS

As we have stated, the most typical manifestations of gout—great toe involvement, tophi, punched-out areas in the roentgenogram—may be missing in a given instance. This need not eliminate consideration of a diagnosis of gout, however. Even when such evidences of gout are present they may not be striking, each suspicion, therefore, must be deliberately investigated. The fact is that in many cases the accurate recognition of gout must be based on familiarity with its characteristic clinical course, which, incidentally, is often charted with great accuracy by the patient himself in his history.

The diagnosis of chronic gout is obvious when joint deformities are associated with the characteristic sequelae of gout—tophi, hyperuricemia and the typical roentgenographic picture. The aim is, however, to determine the existence of gout in its early stages. At this time the manifestations of the disease are transitory, and appropriate treatment may arrest the process, averting permanent damage. During this early phase the characteristic history is. Recurrent attacks of acute arthritis limited to a few joints especially the great toe, with complete subsidence of all signs and symptoms and relatively long intervals between attacks. The history constitutes a guide to a search for some corroborative data such as an increased uric acid content of the blood, or tophi, which establish the diagnosis beyond doubt.

Uric Acid Concentration in the Blood

Many observers have noted the occasional complete dissociation between blood uric acid levels and joint symptoms. Thus, attacks of gout sometimes occur when the blood uric acid concentration is at the upper limit of, or even below normal whereas weeks after the attack hyperuricemia may appear. Moreover, daily variations occur in the serum uric acid concentration in patients even in those who are maintained on controlled diet and medication, they may occur not only in intervals between attacks, but also during an attack. In other cases the serum uric acid concentration remains unchanged throughout the attack of gout.

Jacobson found hyperuricemia, with a concentration of uric acid exceeding 6 mg per cent, in nearly all of an extensive series of determinations in gout. In the majority of them, uric acid values ranged from 7.0 to 14.0 mg per cent. These determinations were carried out on serum derived from blood allowed to clot under oil, employing the technique of Folin (1933). This study suggests that an increased concentration of uric acid in the blood is an invariable accompaniment of established gout. That has not been the experience of many clinicians, including ourselves, who have observed unquestionable cases of gout in which the concentration of uric acid in the whole blood (determined by the Benedict method, 1931) was sometimes well within normal limits. Although a blood uric acid value of over 6 mg per cent confirms a diagnosis of gout, lower values—between 3 and 6 mg per cent—do not always exclude the possibility of gouty arthritis. Such low blood uric acid levels are occasionally encountered in cases in which the diagnosis of gout can be established on the basis of other pathognomonic evidence of the disease.

Other Laboratory Findings

During acute attacks the leucocytes may be moderately increased in number, but the proportion of polymorphonuclear leucocytes is not in

creased whereas the mononuclear cells are. The sedimentation rate is generally normal, it is more likely to be increased during an acute exacerbation in an advanced case.

Therapeutic Tests

When other confirmatory evidence is not at hand, a therapeutic test with full doses of colchicine may establish the diagnosis in an otherwise obscure type of acute arthritis.

Recently Lockie and Hubbard demonstrated striking changes in the symptoms and purine metabolism produced by high fat diets in certain cases. Briefly these observers noted that when patients with gout ingest diets high in fat an acute attack of gout is usually, but not invariably, induced within from two to sixteen days. It is interesting that the clinical manifestations so induced were relatively independent of changes in the uric acid content of the blood. In some patients, such provocative attacks of gout anticipated the rise in blood uric acid concentration, in others, with marked elevation of the blood uric acid the symptoms decreased or disappeared while the hyperuricemia was still pronounced.

In most of the subjects studied the uric acid concentration in the blood increased as the excretion of uric acid in the urine decreased, provided the diet was ingested for a period of days or several weeks. When the high fat diet was replaced by one low in fat and high in carbohydrate, marked improvement but not always complete relief from pain, was obtained, even when the blood uric acid concentration remained high.

Because of their ability to induce acute attacks of gout by feeding diets high in fat Lockie and Hubbard proposed the following test for differentiating gout from various forms of chronic arthritis.

Feed a diet consisting of from 250 to 350 gm. of fat, 50 gm. of protein and from 30 to 50 gm. of carbohydrate for a period of from five to seven days. If within that time pain in the joints has developed, or if existing mild joint pains have markedly increased in severity a diagnosis of gout must be carefully considered. The symptoms that may develop can be promptly relieved by feeding a diet high in carbohydrate and low in fat."

The development or exacerbation of arthritic symptoms following the ingestion of a diet high in fat and low in carbohydrate may, therefore, serve usefully in detecting certain cases of gout which might otherwise elude recognition. It must be remembered, however, that even in known cases of gout a high fat diet is not always effective as a provocative test. Furthermore, an attack of gout may not be induced by such a diet for a period of from ten days to two weeks. Inability to provoke an attack of gout by such a diet does not, therefore, exclude its existence when other evidence suggests its presence.

DIFFERENTIAL DIAGNOSIS

The similarity between the history of gout and rheumatic fever is striking. In both conditions there are recurrent acute episodes of arthritis, with intervals of complete remission. Repeated attacks of rheumatic fever, however, are usually followed by rheumatic carditis, whereas evidence of heart disease is usually absent and chronic nephritis likely to be found in the patient who has had repeated attacks of gout. Again, rheumatic fever usually involves more of the large joints and is accompanied by more fever.

Ordinarily the exclusion of the common type of nonspecific, rheumatoid arthritis is not difficult. It is rare for rheumatoid arthritis to recur more than once or twice without leaving some permanent changes in the joints. As a rule permanent damage occurs during the very first attack of atrophic arthritis. In advanced cases of gout, however, a picture hardly distinguishable from rheumatoid arthritis occasionally develops; this has been thoroughly described by Ludwig, Dennis and Bauer.

TREATMENT

During acute attacks the patient must be at rest with the joints entirely protected from irritation by motion. Cool compresses of magnesium sulphate solution may be used, and when other drugs fail, morphine should not be spared for the relief of pain.

During acute episodes a soft diet, rich in carbohydrate and restricted in fat, and a liberal intake of fluid should be provided. Colchicine can usually be relied upon to control the joint pains. Although the exact mechanism of the pharmacologic action of colchicine is unknown, it is unquestionably one of the most effective drugs for control of the clinical manifestations of the disease. Crystalline colchicine is preferable to the wine or tincture of colchicum, the stability and potency of which may vary. If necessary, and if it is tolerated, colchicine may be administered in doses of $1/120$ grain (0.005 gm.) every one or two hours, for from ten to fifteen doses. The limit of tolerance for the drug is indicated by the development of nausea, vomiting, or diarrhea, at which time further administration must be limited to maintenance amounts, which may vary from $1/120$ to $3/120$ grains daily.

During symptom free intervals the diet should be rich in carbohydrate, moderate in protein, and restricted in fat. It should of course be adequate in minerals and vitamins. The value of massive doses of vitamins, particularly of vitamin B (thiamin chloride), which has been recommended, is not yet established. Dietary regulation designed to eliminate foods espe-

cially rich in purines, and the avoidance of diets high in fat, seems logical though it is doubtful whether rigid restriction of the purine intake is justified during symptom free intervals. The more critically one examines the actual value of extreme purine restriction, the more one is inclined to agree with Lichtwitz that 'the purine free diet stands much higher in the estimation of physicians than of the patients suffering from gout.' During acute episodes of gouty arthritis and in the ensuing several weeks, however, restriction of purine would seem to be advisable.

Foods containing a large amount of purine which should be eliminated from the dietary of the patient with gout

Sweetbreads	Meat extractives
Anchovies	Meat broths
Liver	Sardines
Kidney	Herring
Calves' tongue	Trout
Squab	Pike
Goose	Cod
Turkey	Coffee
Brains	Coca cola
Gravies	

Foods containing moderate amounts of purine which may be permitted for one meal of the day during intervals between acute attacks

Bacon	Caviar
Lamb	Perch
Pork	Salmon
Veal	White fish
Chicken	Haddock
Mutton	Oyster
Sausage	Crab
Beef	Lobster

The diet may be selected from the following

Milk	Tapioca
Eggs	Gelatin
Cheese	Fruits of all kinds
Cream (in moderation)	Vegetable soup (made without meat)
Butter (in moderation)	Cereals (except whole grain)
Nuts	Caffeine free coffee
Sugar and Sweets	Bread (except whole grain)
Weak Tea	Honey

Cocoa	Jelly
Rice	Jam
Macaroni	
Marmalade	

Vegetables of all kinds are permitted except the following

Lentils	Beans
Mushrooms	Kohlrabi
Peas	Spinach

Radishes water cress, paprika mustard, relishes, horse radish, catsup and other spices and condiments should be largely avoided

The patient afflicted with gout should adopt favorable hygienic measures including moderate exercise, but avoiding physical fatigue and nervous stress. He will naturally do well to eat moderately. Obesity is a distinct detriment and should be prevented or eliminated. Although alcoholic beverages are thought to be distinctly harmful, it is not proved that they need be rigorously excluded. Focal infection may play a contributory role and is better eradicated.

The value of salicylates—either sodium salicylate or acetylsalicylic acid—in aiding the excretion of uric acid is now definitely established. They may be administered in doses of 40 to 80 grains daily, for three or four days in the week, and may serve to prevent, or at least postpone, acute exacerbations of gouty arthritis.

Patients who are subject to several attacks of gout each year may be given two or three doses of 1/120 grains of colchicine daily, two or three days a week. Salicylates, in the dosage indicated above, may be employed during the other days.

Cinchophen preparations had been recommended for the alleviation of pain. They undoubtedly encourage the excretion of uric acid, so that following their administration hyperuricemia may be perceptibly reduced. The toxicity of cinchophen and the dangers associated with its use are, however, real deterrents. Even small doses of the drug may lead, without appreciable warning to fatal liver damage. Although such toxicity apparently occurs only in those who have some inherent idiosyncrasy to this drug the danger of poisoning is so real that ordinarily the drug should not be used when there is an effective substitute. Colchicine and salicylates are fully as effective as cinchophen in the treatment of gout. We believe, therefore, that the use of cinchophen preparations is not justifiable. If the drug is used it should be discontinued upon the appearance of the earliest manifestations of toxicity, such as nausea, other digestive symptoms, urticaria, pruritus, or jaundice.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527]

CHAPTER XXIII

GONOCOCCAL ARTHRITIS

Gonococcal arthritis is so frequently polyarticular that it may easily be taken for nonspecific atrophic arthritis, particularly by those who associate the idea of gonococcal joint infection with monarticular involvement. The diagnosis of this type of arthritis is now more important than ever before, since, if treatment is instituted before permanent damage has occurred, curative measures are available for all but the most resistant cases.

Although it is most common in young adults and occurs somewhat more frequently in men than in women, gonococcal arthritis may appear at any age in either sex and it does not exempt individuals of any social status.

PATHOLOGY

The pathologic features of gonococcal arthritis are essentially those seen in any infectious joint disease. The brunt of the infection falls upon the synovial membrane and periarticular structures. There is edema of the synovial lining, congestion of blood vessels in the underlying connective tissue, and infiltration with leucocytes, chiefly polymorphonuclear cells, throughout. The synovial membrane may proliferate into a pannus which projects into the joint cavity. If the inflammatory process is severe, the lining cells of the synovial membrane may actually become necrotic and desquamate, leaving raw, ulcerated surfaces of granulation tissue covered with fibrin and greenish yellow pus. The synovial fluid is increased in amount, and generally cloudy, it may be frankly purulent. The knee joints are apt to develop the largest synovial effusions. If the synovial lining becomes necrotic and ulcerated, gonococci may spill over into the joint effusion, otherwise, the synovial fluid is apt to be sterile, though cloudy from the presence of polymorphonuclear leucocytes.

If the infection is not arrested or burned out early, the joint is seriously damaged, the cartilage is destroyed through proliferation of the infected synovial pannus and is digested by the tryptic activity of the leucocytes in the synovial exudate. Apparently, destruction of cartilage occurs more readily when the exudate is purulent, it is likely to be less severe if the purulent exudate is diluted by relatively large amounts of synovial fluid.

Finally, fibrosis may occur, entirely obliterating the joint cavity by a mass of fibrous tissue. If the cartilage has been partially or wholly destroyed, its remnants or the underlying bone may become involved in the mass of fibrous tissue, a stiff, ankylosed joint resulting. Thus true bony ankylosis may eventually supervene.

The general pattern of the inflammatory process and pathologic change within the joints in gonococcal arthritis is not at all unlike that which occurs in nonspecific atrophic arthritis, except that the pathologic change in gonococcal arthritis is more purulent in character and, if not treated, may lead to earlier destruction of the joint.

CLINICAL MANIFESTATIONS

Gonococcal arthritis is generally ushered in abruptly as an acute periarthritic arthritis, sometimes monoarticular, but more often affecting many joints at one time. When the arthritis is polyarticular it may be indistinguishable in its general aspects from an acute nonspecific atrophic arthritis. The pain in gonococcal arthritis is likely to be very severe, however, even when the patient is at rest.

A frequent mode of onset is with polyarticular affection, severe joint pains and stiffness everywhere, lasting a day or two, and subsequent localization to one or two joints, which become exquisitely painful. The knees, ankles, and wrists are affected most commonly, but any joint may be involved. The joints become markedly swollen, tender, sometimes red and hot. There is, of course, marked muscle spasm about affected joints. Jarring or the slightest passive motion produces agonizing pain. Therefore, the patient guards the affected joints against movement or palpation with the greatest anxiety. Muscle atrophy, of marked degree, becomes evident rather quickly, frequently within a week or two.

Severe tenosynovitis, particularly about the wrists and ankles, is frequently an associated symptom. Acute tenosynovitis may occur without frank arthritis and is a common manifestation of gonococcal infection. When no other cause for such tenosynovitis is evident, the possibility of existing gonorrhea should always be considered.

A premonitory chill, such as occurs so often preceding various types of septic arthritis, is unusual in gonococcal arthritis. In any event, the joint involvement is accompanied by evidence of a systemic infectious process, with fever, sweating, leucocytosis, and rapid sedimentation of erythrocytes. The fever is generally not as high as might be expected from the severity of the arthritis.

In most cases the chronologic relationship to a recent gonococcal infection of the genito-urinary tract is clear, the arthritis appearing within from

ten to twenty days after the onset of the gonorrhea. Obviously this sequence is noted more readily in men than in women. The latter may have been unaware of the initial gonorrheal infection except to the extent that they may recall the existence of leucorrheal discharge or dysuria which was possibly attributed to bladder inflammation. An etiologic relationship may be less clear in patients with acute gonococcal arthritis occurring months or even years after an attack of genital gonorrhea. When the appearance of gonococcal arthritis is so long delayed its development is likely to have been precipitated by sexual excesses perhaps by a debauch of drinking by vigorous prostatic massage by manipulation of infected pelvic organs during surgical operations and in some cases by pregnancy. Although a history of a recent attack of gonorrhea is an important diagnostic lead when gonococcal arthritis is suspected denial of venereal infection does not dismiss the possibility of its existence.

In some cases the occurrence of conjunctivitis either before or coincident with the appearance of the arthritis or the presence of iridocyclitis arouses the suspicion of gonococcal arthritis when other leads may be missing.

DIAGNOSIS

Acute gonococcal arthritis may be preceded by a sore throat or upper respiratory infection which suggests misleadingly the possibility of acute rheumatic fever or acute nonspecific arthritis. In a small proportion of cases the onset is less acute than usual again suggesting the possibility of an atrophic arthritis. Gonococcal arthritis developing as a result of gonorrhea of long duration and relative latency is apt to develop insidiously and to progress in a more or less subacute or chronic form resembling in every respect the mode of onset and progression of the more usual atrophic arthritis.

The diagnosis of gonococcal arthritis must be made conclusively; a presumptive diagnosis is not enough. The history and the clinical examination alone do not always suffice. A close chronologic relationship between a recent acute attack of gonorrhea the finding of an active genital infection and the typical onset and manifestations of gonococcal arthritis are strongly suggestive of the diagnosis. But to establish it beyond doubt further study is necessary and frequently yields conclusive data.

Laboratory Aids

The blood count is of little importance from the standpoint of confirming the diagnosis because it reveals only those changes associated with an active infective process. Nor is the increased rate of sedimentation of diag-

nostic value. The examination of synovial fluid obtained from an affected joint is, however, of great importance.

Synovial Fluid

In gonococcal arthritis the total cell count varies between 1500 and 150,000 per cubic millimeter. The count may go as high as 200,000 cells. Higher cell counts occur in fluids from which the gonococcus is obtainable on culture, and lower counts in those with negative cultures. The proportion of polymorphonuclear leucocytes is likely to exceed 75 per cent and is again, higher in infected than in noninfected fluids. The complement fixation test on the fluid is positive in about 75 per cent of cases of gonococcal arthritis.

The gonococcus should be looked for, it may be detected either in direct smears or on culture in about one third of the cases. Cultures are more desirable, for when the organism can be detected in direct smear, it is practically always recovered on culture, but the reverse is not true. A satisfactory means of isolating the gonococcus is to streak ascitic agar plates with the pus or synovial fluid as soon as it is obtained. They are then placed in anaerobic jars, which are sealed and incubated, after carbon dioxide gas has been blown into them.

It must be remembered, however, that the gonococcus may be recovered in cultures in only about one third of the cases subsequently proved to be gonococcal in nature. This means that the inability to demonstrate the organism does not rule out existence of this disease.

Gonococcus Complement Fixation Test

As already stated, this test on synovial fluid is positive in about 75 per cent of cases. With blood serum the test yields a positive reaction in about 80 per cent of proved cases of gonococcal arthritis. Warren, Hinton and Bauer pointed out that in those cases in which the history is consistent with the diagnosis of gonococcal arthritis a positive complement fixation test on the patient's serum will be correct in about 90 per cent of the cases. Obviously this test offers great assistance in establishing the diagnosis. It must be remembered, however, that in about 20 per cent of the cases a negative reaction occurs but does not rule out gonococcal infection. An excellent statistical analysis of this test as a diagnostic aid in the study of arthritis is offered by Warren, Hinton and Bauer (1937). Their paper includes detailed description of the actual technique of performing this test.

Manifestations in the Roentgenogram

In general, the roentgenographic manifestations in gonococcal arthritis are those of an atrophic, periarthritic type of joint disease. The chief char-

actenstics are the presence of a sharply outlined effusion associated with periarticular swelling which is much less pronounced however than that seen in pyogenic arthritis. Shortly afterward a sharply circumscribed local decalcification about the affected joints develops. Still later there is narrowing of the joint space resulting from cartilage destruction and a more general decalcification. When ankylosis exists the roentgenogram is not likely to reveal the dense bony ankylosis seen in nonspecific atrophic arthritis but rather calcareous striae bridging the joint. Bone destruction is minimal in degree or altogether absent contrasting with the findings in advanced tuberculous or septic arthritis. Nor is there evidence of much hypertrophic bony change about areas of cartilage destruction.

The diagnosis of gonococcal arthritis depends then on study of the following factors: (1) The clinical course of the disease; (2) the finding of gonorrheal infection in or the isolation of the organism from the genito-urinary tract; (3) detection of gonococci in direct smears or cultures of synovial fluid; and (4) eliciting of a positive gonococcus complement fixation test on the fluid or blood.

Obviously one need not expect to find all of these factors in every case. Repeated attempts should always be made to isolate the organism by culture from synovial fluid, from urethral and prostatic secretion in the male and from the vagina, cervix and Bartholin's and Skene's glands in the female. In the latter, positive cultures are more likely immediately prior to or following menstruation.

The complement fixation test is useful as suggestive evidence when the organism cannot be isolated. Its principal value is corroborative; unsupported by other evidence it is not diagnostic. Indeed synovial fluid which harbors many gonococci is likely to yield a negative complement fixation test and vice versa. It is also to be remembered that although the complement fixation test on the blood may turn positive as early as the first week of the disease, such a reaction may be delayed for several weeks. Hence a single test shortly after the onset of the disease is not conclusive; it may have to be repeated to establish the diagnosis of gonococcal arthritis.

DIFFERENTIAL DIAGNOSIS

Examination of joint fluid may serve not only to establish the diagnosis of gonococcal arthritis but also to exclude other types of acute suppurative arthritis. The latter may be easily confused with gonococcal arthritis particularly when an acute monoarticular disease exists. Discovery of the specific organism in the fluid aspirated from the affected joint is the definitive factor in differential diagnosis.

The possibility of acute rheumatic fever may be suggested by the initial

symptoms of widespread infection, with swelling and soreness of many joints, and subsequent localization of the arthritis to one or two of them. It is especially confusing if the arthritis is preceded by an acute upper respiratory infection, and, in such cases, the differential diagnosis may be impossible to establish on clinical grounds alone. Study of the synovial fluid, complement fixation tests, and examination of the genitourinary tract may be the only means of settling the issue.

We have already stated that gonococcal arthritis may have an onset and follow a course much like that of ordinary nonspecific, atrophic arthritis. The original gonococcal genital infection may have occurred long before the onset of the arthritis and may, in fact, have become clinically quiescent before the arthritis began. The synovial fluid may be of little aid in differential diagnosis if the gonococcal arthritic process has attained a stage of chronicity. Analysis of the history and of the course of the disease, study of the genitourinary tract and the complement fixation test on the blood may establish the diagnosis of gonococcal arthritis. If such study is not conclusive, and serious suspicion of gonorrhea still exists, a therapeutic test with sulfanilamide or induced fever may be useful. These measures, however, do not cure all cases of gonorrhea. Certain strains of gonococci are resistant to even the highest ranges of therapeutic fever and massive doses of sulfanilamide, under such circumstances the therapeutic test could be misleading.

Acute gout may resemble gonococcal arthritis in some respects. The blood uric acid concentration, study of roentgenograms, a search for tophi, the complement fixation test on the blood, and examination of the synovial fluid establish the diagnosis.

Acute inflammation of the tendo achilles or of the tendons about the wrist occurs not infrequently as a result of gonococcal infection. Teno-synovitis may or may not be associated with frank arthritis.

TREATMENT

The immediate consideration in the treatment of gonococcal arthritis is relief of acute pain. This can be accomplished through immobilization of affected joints either by splinting or traction. Not only is pain relieved, but the hazards of extensive damage, particularly destruction of cartilage, are in this way averted.

Neither the local application of short wave diathermy or other forms of heat, nor hydrotherapy, can be depended upon for complete eradication of the infection.

Antigonococcus vaccine, filtrates, and serums have been employed with

variable success. The therapeutic gains with these agents do not compare with those achieved with fever or chemotherapy.

Fever and chemotherapy with sulfanilamide offer the most reliable methods for the control of gonococcal arthritis. In the vast majority of instances both the primary focus as well as its metastatic effects are eliminated by these means. Of the two, chemotherapy appears to be the more reliable and most easily applied.

In contrast to the discouraging results obtained with fever therapy in the management of atrophic arthritis, its effect in gonococcal arthritis is veritably spectacular. Fever therapy offers one of the surest ways of attaining cure in gonococcal arthritis. The treatment may be administered in one session, extended over many hours, or in a series of two to five shorter sessions. The total exposure to fever and the height of the temperature determine the final result. The treatment may be administered in electrically heated, insulated cabinets, in carefully controlled hot water baths, or by the injection of fever-inducing substances such as dead typhoid bacilli. The heated cabinet (hypertherm) is the most satisfactory.

Fever therapy, however, cannot be entered into lightly. It carries a very definite risk, and fatalities occasionally occur even when it is administered by the most experienced technicians. It demands more than the mere application of the appropriate apparatus. Patients must be examined most thoroughly before being subjected to fever therapy. The cardiovascular system must be competent; chronic alcoholic addicts, the aged, and the debilitated do not tolerate fever therapy well. While the treatment is being administered, the patient should be under the constant supervision of a specially trained nurse. A physician also must be in close attendance to be alert to any unfavorable reactions.

Fever therapy is of course most efficacious in the earliest stages of the disease, when cure with full restoration of function may reasonably be expected. It cannot be curative in old, burned-out cases with ankylosis. It may also fail when the strains of gonococci are too resistant to destruction by the ordinary, safe therapeutic ranges of fever.

SULFANILAMIDE

Recent reports reveal that the effectiveness of chemotherapy with sulfanilamide exceeds even that of fever therapy in gonococcal arthritis. Naturally, the best results here too are secured in the early, acute cases, before irreparable damage has occurred. Bauer and Coggeshall have shown that when sulfanilamide is administered in large doses the infected synovial fluid can be rendered sterile within forty-eight to seventy-two hours, during which time striking clinical improvement also appears. The erythrocyte sedimentation rate is consistently reduced to normal. In about half the

cases the gonococcal complement fixation test, if positive before treatment, tends to become negative within two months, this test may never become positive if treatment is instituted early enough

From a fairly large experience with sulfanilamide Bauer and Coggeshall have arrived at the following therapeutic schedule

"The daily intake of fluid is usually maintained at 2,000 cc The dose of sulfanilamide is calculated in the following manner $\frac{3}{4}$ grain per pound of body weight, providing the total dose does not exceed 120 grains, or 8 gm This calculated dose represents the amount to be given every twenty four hours

Knowing the calculated dose, the drug is then administered orally in one of two ways (1) half the calculated dose is given initially and again in four hours and then one sixth of the calculated dose is given every four hours day and night, (2) one sixth of the calculated dose is given initially and every four hours When the drug is given according to the first schedule, a blood sulfanilamide level of from 10 to 15 mg per cent will result, whereas adherence to schedule 2 will allow for the maintenance of a blood sulfanilamide level of between 5 and 10 mg per cent "

These writers emphasized the importance of maintaining constantly a sufficiently high concentration of sulfanilamide in the blood, at a level of from 10 to 15 mg per cent, a state which may be attained if large enough doses of the drug are administered regularly, as described Small doses may not only fail to effect a cure but may actually increase the resistance of the organism to subsequent destruction by chemotherapy

The use of sulfanilamide is frequently accompanied by toxic manifestations (usually mild, possibly severe, occasionally serious) Cyanosis of some degree occurs in practically all patients receiving large doses of the drug The administration of sodium bicarbonate with sulfanilamide has been suggested to minimize the danger of acidosis Severe hemolytic anemia, leucopenia to the point of agranulocytosis, febrile reactions, and skin reactions may develop Obviously, then, a patient under treatment with sulfanilamide should be observed closely for the appearance of toxic reactions, and necessary steps must be taken to combat any toxemia that may develop Most of these complications can be controlled readily by discontinuing the use of the drug and giving large amounts of fluids If severe anemia or agranulocytosis develops, transfusions may be necessary

Of all forms of therapy for gonococcal arthritis, including induced fever, Bauer and Coggeshall were most impressed with the results achieved by chemotherapy with sulfanilamide

Inability to achieve satisfactory cure of gonococcal arthritis with sulfanilamide should arouse suspicion of inadequate dosage If blood sulfanilamide determinations establish this to be the case, the dosage should be increased

to the required level, at which time satisfactory results may be secured. However, a small percentage of gonococcal infections are not cured even by large doses of sulfanilamide. In certain of these instances one is probably dealing with "sulfanilamide resistant" gonococcal strains. In such an event it is logical to try the effect of sulfapyridine, of fever therapy (if it is available), or of a combination of fever therapy and sulfanilamide.

The place of sulfapyridine or of other sulfanilamide derivatives alone in the treatment of gonococcal arthritis is not yet established.

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CHAPTER XXIV

TUBERCULOUS ARTHRITIS

Tuberculosis of joints is as insidious a disease as is tuberculosis of the lungs—and equally as destructive. Hence the importance of early diagnosis. Although usually monarticular, it may affect multiple joints. On the other hand, monarticular arthritis, clinically simulating tuberculosis, may on surgical and pathologic investigation prove to be nontuberculous.

Tuberculosis occurs most often in joints that are subject to the greatest trauma in the ordinary course of living. Thus, the spine, hips, and knees are most commonly affected. Although it is a disease of children rather than of adults, tables of age and sex incidence must not be taken too literally. A chronic inflammatory process in any joint, at any age, may be tuberculous.

PATHIOLOGY

It appears that trauma may act as a predisposing factor, permitting localization of the tubercle bacilli which produce the tubercle, essentially a chronic granulomatous reaction in tissues. The differences in the lesions seen in different tissues are conditioned only by the nature of the tissue affected.

The tubercle bacillus attacks synovial tissue primarily in some cases, and the epiphyseal ends of the bone in others. The primary pathologic change may therefore be confined to one or the other of these sites. From a clinical standpoint a knowledge of these primary sites of invasion is important. It indicates that joint tuberculosis affecting the synovia may exist when the roentgenogram shows no sign of bone destruction. Eventually, however, both of these sites are involved regardless of where the process began.

The synovia of a tuberculous joint becomes thickened and gray, roughened by glistening white or grayish tubercles. The joint usually contains a yellowish, turbid fluid and the cartilage becomes overlaid with highly vascular, pink granulations, which constitute the synovial pinnus. In the subchondral, epiphyseal ends of the bone, destruction of cancellous bone develops, the overlying cartilage becomes yellow and gradually necrotic. However, the pathologic process is not altogether a destructive one, an

attempt to wall it off, and even to regenerate bone, is always in evidence. In this direction, though, nature's attempt is none too effective, the destructive process generally maintains the upper hand.

CLINICAL MANIFESTATIONS

The patient with joint tuberculosis is rarely critically ill when the physician first sees him. Generally he complains merely of swelling of a single joint with pain that is for the most part, deceptively mild. Even with ordinary use of the joint, pain is rarely severe. There may be muscle spasm of varying degrees. If the patient has been at rest, muscle spasm may not be demonstrable until forced motion is attempted. In the lower extremities, particularly with involvement of the hip, pain on walking, alleviated by rest, and a persistent limp may for a long time remain the only complaints. Such pain of hip disease may be referred to the knee.

Although tuberculosis of the spine is not as prevalent as it was years ago, it must not be forgotten as a possible cause of pains in the back. The spine is, in fact, the most common site of tuberculous arthritis. It affects children much more often than adults, and the thoracic segments are most frequently involved.

The bodies of the vertebrae and the intervertebral disks suffer the brunt of the damage. Since the anterior part of the vertebral bodies are involved to the greatest extent, softening of these areas and their ultimate collapse produces the characteristic angulation (kyphosis) of the spine so characteristic of advanced, neglected cases.

Tuberculous spondylitis may produce pain, soreness, and limitation of motion confined to the area affected, or it may induce pains in more remote regions. Thus, pain may be referred to the chest or abdomen with involvement of the dorsal vertebrae, and to the legs with involvement of the lumbar spine. Such symptoms (especially in children) should lead to search for other signs of spinal disease, such as local tenderness, muscle spasm, limitation of mobility and kyphosis. By considering the possibility of tuberculous spondylitis in its early stages, before obvious roentgenographic manifestations appear, we may prevent deformity and abscess formation, which need not occur.

Not infrequently a history of joint injury is related. The joint swells after such trauma, presumably a traumatic synovial effusion occurs. The swelling may then recede during rest, but recurs repeatedly and persistently with activity. In some patients, the swelling never disappears. As the disease progresses and the synovia thickens, more and more of the swelling is attributable to synovial hyperplasia rather than effusion. In joints that are deeply situated anatomically, the swelling may not become evident clini-

cilly until late in the disease if at all. As a rule tuberculous joints are not red and are not hot to the touch. Neither are they so sore on pressure as the swelling would lead one to expect. The swelling may assume a fusiform appearance and in such joints as the knee and ankle where swelling is easily demonstrable it may be doughy on palpation.

In tuberculous arthritis the constitutional manifestations vary greatly in severity. Children show more labile reactions with fever and malaise but even in adults there may be low grade fever, anorexia and weight loss. Occasionally high fever, prostration and all the local signs of an acutely inflamed joint may appear. Visceral tuberculosis in the lungs or elsewhere may coexist but frequently it is not demonstrable clinically.

The affected joint may be limited in motion merely by muscle spasm. Later motion may be limited by actual obstruction resulting from the destructive process within the joint and the proliferating pannus. In late cases the joint is often palpably unstable with abnormal side to side motion resulting from destruction of the stabilizing ligaments.

DIAGNOSIS

We have already stated that the possibility of tuberculous arthritis can not be dismissed when more than one joint is involved, that the elderly patient is not necessarily exempt and that we must not ignore the possibility of its existence in patients with histories of trauma. Occasionally what is at first a traumatic internal derangement of the knee develops into a frankly destructive tuberculous arthritis. In short the clinical picture is so varied that any chronic joint involvement may be tuberculous. Chronic joint disease associated with demonstrable cold abscesses and perforating sinuses is practically always tuberculous in nature.

Röntgenographic Findings

As a rule tuberculous arthritis is fairly advanced before bone destruction becomes evident in the roentgenogram. In earlier cases the roentgenogram may reveal only thickening of the capsule and a fairly typical ground glass loss of density of the bone ends. In early synovial tuberculosis the most prominent features in the roentgenogram are a dense sharply defined of fusion slight local decalcification of the bone ends little or no periarthritic swelling but marked soft tissue atrophy. There may be slight narrowing of the joint space but there is no destruction of bone and proliferative bone reaction is altogether absent.

When the tuberculous process begins primarily in the bone as occurs more often in adults than in children focal decalcification or destruction of bone in the epiphysis may develop very early. When such areas of bone

destruction occur, the surrounding bone also becomes decalcified. In more advanced stages, the entire width of subchondral bone and cartilage may be penetrated by the inflammatory pannus, which extends into the joint cavity. With destruction of the cartilage the joint space becomes narrowed or obliterated. Effusion of fluid may also develop, but it is generally less marked than in primary synovial tuberculosis. The chronologic order of appearance of these various factors, and the degree of change, are important considerations upon which the experienced roentgenologist bases his diagnosis. With such experience and correct interpretation of the roentgenographic findings, an accurate diagnosis of tuberculous arthritis may be made quite early.

Strictly speaking, a diagnosis of tuberculous arthritis can be made only by demonstrating the organism in joint fluid or tissue, or by demonstrating the typical tubercle in tissue histologically. Despite the aseptic technique available today, it is sometimes more practical, from the point of view of successful treatment, to act on a presumptive clinical diagnosis of tuberculous arthritis than to penetrate the joint for confirmatory evidence.

Confirmation of the tuberculous nature of arthritis may be secured by means of roentgenograms, by proving the existence of associated visceral tuberculosis, and particularly, by the results of guinea pig inoculation of joint fluid, or by biopsy. The latter is the most reliable. A negative Mantoux test in an adult is generally suggestive of the absence of tuberculous infection.

Synovial Fluid

The total count may vary between 5,000 and 10,000 cells per cubic millimeter. In tuberculous arthritis there is likely to be a greater proportion of lymphocytes and monocytes than in fluids with comparably high total cell counts obtained from pyogenic joints. In any event, guinea pig inoculation of the suspected fluid should be performed in every case of tuberculous arthritis in which fluid has been aspirated. Aspiration of such joints must be performed with meticulously aseptic technique, for tuberculous joints are highly susceptible to secondary invasion by pyogenic organisms. For this reason, too, repeated aspirations are to be avoided.

TREATMENT

The first consideration in the treatment of tuberculous arthritis is the matter of conservative, nonsurgical, versus surgical, management. Realizing the fact that the inherent capacity for healing in tuberculous arthritis is extremely limited, it follows that in most cases the cure depends upon attaining surgical arthrodesis of the affected joints. The success of surgical

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treatment depends entirely on producing a thoroughly ankylosed joint. Although this therapeutic approach offers the quickest way to cure and therefore to earliest rehabilitation of the individual, it also means leaving the patient with a stiff joint. Regardless of the desirability of preserving joint function, surgical fusion sooner or later becomes necessary in most cases of tuberculous arthritis. Even at the expense of a functionally useless joint, this may be the only way of attaining a cure that has any promise of permanence. Certainly the patient who has suffered destruction of cartilage and epiphyseal bone is not apt to escape surgical arthrodesis, for in no other way is such a joint likely to be healed.

In cases of early synovial tuberculosis, particularly in children, the matter of conservative treatment may be considered more seriously and hopefully. This does not imply that many such cases can be cured by conservative management. To be sure, in many of these cases progression of the arthritis may be stopped and perhaps even quiescence of the process may be attained, but in most of these the tuberculous process, instead of remaining quiescent, flares into renewed activity when the joint is again subjected to the trauma of even physiologic use. However, prolonged immobilization of the joint, with or without traction, and appropriate systemic management of the patient may yield an occasional recovery with useful function. That is worth aiming for.

Regardless of the type of local treatment chosen, systemic treatment of the patient must always be an integral part of therapy in tuberculous arthritis. Provision for systemic rest, adequate nutrition, fresh air and sunshine is essential for the treatment of any type of tuberculosis, including tuberculous arthritis. These measures must be employed regardless of whether the patient is treated medically or surgically.

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CHAPTER XXV

SOME LESS COMMON FORMS OF SPECIFIC INFECTIOUS ARTHRITIS

ACUTE SUPPURATIVE ARTHRITIS (PYOGENIC ARTHRITIS, PYARTHROSIS)

Consideration must be given here to acute suppurative arthritis because so many cases of this condition are mistaken for either acute gonococcal arthritis, rheumatic fever, or atrophic arthritis. The importance of early diagnosis of acute suppurative arthritis cannot be overestimated. Treatment of this condition differs radically from that of most other types of joint disease. Unless drainage of the infection is instituted early, not only the joint but the patient's very life may be jeopardized.

It is unnecessary to review details of the pathologic and clinical manifestations of acute suppurative arthritis, they are essentially those of any acute infectious joint disease.

An inflamed, painful, large, swollen joint with effusion may be the seat of any type of infectious process. The diagnosis of acute suppurative arthritis cannot be established on the basis of clinical findings alone. Only aspiration and examination of fluid from the joint can clinch that diagnosis. In suppurative arthritis the fluid is distinctly purulent, with many polymorphonuclear leucocytes and fibrin. The specific organism can usually be demonstrated both by direct smears and by culture.

Röntgenographic examination in the early stages of the disease may reveal little change of diagnostic value. Even in more advanced stages the narrowing of the joint space resulting from cartilage and bone destruction is not characteristic of this process alone, and may resemble many other types of severe, acute joint inflammation.

Treatment. The successful management of this type of arthritis depends upon its early recognition. The basic principle in treatment is adequate drainage, either by repeated aspiration or incision. In the later stages the treatment aims to control bone destruction or, if that has already developed, to prevent ankylosis or to favor fusion in as favorable a position as possible.

Systemic treatment to combat the toxemia of sepsis is unnecessary if early diagnosis is made and appropriate treatment is instituted promptly

PNEUMOCOCCAL ARTHRITIS

Pneumococcal arthritis generally represents either a complication in the course of lobar pneumonia or a metastatic effect of pneumococcic septicemia. This condition is relatively uncommon. Its manifestations are those of acute suppurative arthritis and the diagnosis is established by detection of the specific organism in fluid aspirated from the joint. The occurrence of acute monarticular arthritis in the course of a known pneumococcic infection would of course arouse suspicion of the possible genesis of the arthritis.

Treatment consists of aspiration or surgical drainage of the affected joint. Chemotherapy with sulfanilamide or sulfapyridine as well as specific serum therapy is logical in appropriate cases.

ARTHRITIS OF MENINGOCOCCUS (CEREBROSPINAL) MENINGITIS

Meningococcus meningitis may be ushered in by acute polyarthritis resembling rheumatic fever in many respects. This type of arthritis generally occurs in cases of severe meningococcus infection and is believed to be caused by hemorrhage into the synovial sac (Herrick).

Another and quite different form of meningococcal arthritis occurs. It is generally monarticular, usually affecting the knee or some other large joint and apparently represents a true metastatic infection with meningococci. This type of joint involvement which generally occurs after the fourth or fifth day of the meningitis is characterized by considerable swelling and joint effusion. The joint fluid is purulent, sometimes hemorrhagic. Meningococci may frequently be recovered on culture.

Although this type of meningococcal arthritis may pursue a rather protracted course, recovery with good function generally results.

Treatment with sulfanilamide alone or in conjunction with antimenococcic serum may be employed.

ARTHRITIS OF SCARLET FEVER (SCARLATINAL RHEUMATISM)

Nonsuppurative acute periarticular arthritis occurs occasionally in the course of scarlet fever. Boyd found its incidence to be less than 2 per cent. It generally appears between the fourth and the tenth day of the scarlet

fever. The process consists essentially of acute synovitis with serous effusion into the joint and in some cases periarticular swelling and thickening. Most frequently it affects the wrists, ankles, knees, feet and small joints of the hands. There is a tendency to symmetrical and sometimes migratory joint involvement with new joints affected as others clear. In this respect as well as in the general character of the clinical manifestations the arthritis of scarlet fever may resemble acute rheumatic fever very closely. In fact it has been suggested that scarlatinal rheumatism is really rheumatic fever precipitated by the hemolytic streptococcal infection of scarlet fever.

The condition is self limited. It may last several days to a week but finally clears without residual damage.

Treatment is essentially symptomatic and consists of rest and protection of the involved joints, administration of salicylates internally and external application of methyl salicylate.

Polyarticular suppurative arthritis may also develop in the course of scarlet fever constituting essentially the metastatic joint lesions of hemolytic streptococcal septicemia. The joint fluid is then cloudy, fibrinopurulent; the streptococcus hemolyticus can usually be cultivated from it.

The treatment of this type of suppurative arthritis may include in addition to surgical drainage the use of blood transfusions, scarlet fever antitoxin and sulfanilamide.

SYPHILITIC ARTHRITIS

Syphilitic arthritis (not the Charcot joint) may assume almost any form and may therefore resemble every other type of chronic arthritis.

Mild polyarticular arthritis, generally not very painful, associated with moderate swelling, slight effusion of fluid and some tenderness may occur during the secondary stage of syphilis. Other common manifestations of secondary syphilis are found in conjunction with the arthritis and the blood Wassermann reaction is generally positive.

During the tertiary stage of syphilis a monoarticular type of arthritis, generally affecting the knees, may develop insidiously and pursue a mild chronic course. Although there may be considerable swelling and thickening of synovia and periarticular structures and some effusion of fluid, pain is not severe and limitation of joint motion is relatively negligible.

The possibility of tuberculous arthritis may be suggested by the monoarticular distribution of the arthritis. However the differential diagnosis should not be difficult. The findings in the synovial fluid are helpful. In syphilitic arthritis the total cell count varies between 1,000 and 5,000 cells, the majority of which are either lymphocytes or monocytes, not polymorphonuclear cells. The Wassermann reaction on the fluid is generally

positive and the results of guinea pig inoculation negative. The blood Wassermann reaction is also likely to be positive. Even in syphilitic arthritis of long standing the roentgenogram is likely to show very little cartilage or bone destruction but a significant periostitis may possibly be found adjacent to the affected joint. The diagnosis is moreover confirmed by the result of antisyphilitic treatment. A positive blood Wassermann test alone is not sufficient for the diagnosis of syphilitic arthritis since ordinary atrophic arthritis may occur in patients who are syphilitic. Although such patients may improve upon treatment of the syphilis the arthritis is likely to follow its usual course.

ARTHRITIS OF BRUCELLOSIS

One of the complications of brucellosis may be arthritis which may be mild or severe, monoarticular or widespread and closely resemble ordinary atrophic arthritis. If the joint disease appears early its constitutional manifestations may divert attention from the primary brucella infection.

Migratory polyarthritis may occur which may be mistaken for rheumatic fever. In some cases massive effusions into joints may develop but the arthritis may be relatively painless.

The diagnosis rests largely upon agglutination and intradermal tests with the appropriate antigen. The interpretation of the results of these procedures requires consideration of many factors. The diagnosis is however important for this condition may respond favorably to treatment with the specific serum or vaccine, fever therapy or sulfanilamide.

ARTHRITIS OF HAVERHILL FEVER (ERYTHRA ARTHRITICUM EPIDEMICUM)

Haverhill fever (first described in 1926 as an epidemic disease characterized by abrupt onset with chills, fever, vomiting and a morbilliform eruption) has as one of its most characteristic manifestations a polyarticular arthritis which generally appears on the fifth to the seventh day of the illness. The arthritis may be only mild but in other cases it is severe associated with marked redness, swelling and effusion of fluid into the joints. In such cases an extreme degree of disability from the arthritis may result but even then recovery of joint function ensues in most cases after a period of weeks or months. A specific highly pleomorphic organism (*Haverhillia multiformis*) has been isolated both from the blood of patients and from joint fluid. The condition may be transmitted either by rat bites or by infected milk. This condition must not be confused with the Japanese rat bite fever (Sodoku) which is caused by an entirely different type of organ-

ism a spirochete *Spirillum minus*. In Sodoku arthritis is absent although muscular pains and arthralgias may occur.

ARTHRITIS ASSOCIATED WITH LYMPHOGRANULOMA VENEREUM

Periarticular arthritis occurring with lymphogranuloma venereum has been described. Multiple joints are affected but there is a particular predilection for the knees, ankles and wrists. Dawson and Boots have frequently noted simultaneous involvement of both knees or both ankles. The arthritis generally manifests itself as a chronic indolent type of joint disease with a tendency to effusion into the joints, sometimes to intermittent hydrops and a marked tendency to relapse. Occasionally the condition may manifest itself with acutely swollen, painful, tender joints. The joint fluid is serous in character, sterile and never purulent.

The disease follows a variable course; it may clear up spontaneously in the course of several weeks or it may pursue a chronic, intermittent course for months or years. Despite the fact that the arthritis may persist for weeks or months, the pathologic change remains confined to periarticular swelling and joint effusion without any tendency to bone or cartilage destruction. In each of the 24 cases observed, Dawson and Boots found a positive Frei skin reaction in addition to other evidence of infection with granuloma venereum. However, neither the virus nor the Frei antigen could be demonstrated in the synovial fluid.

In suspected cases, treatment with preparations of antimony (particularly *fuadin*) or by the administration of Frei antigen intravenously or sulfanilamide should be tried.

ARTHRITIS ASSOCIATED WITH ULCERATIVE COLITIS

Ulcerative colitis of the type presumably caused by Barger's diplococcus is complicated by joint involvement resembling atrophic arthritis in nearly 5 per cent of cases. Treatment is directed not only to the condition in the joints but also to the underlying disease.

ARTHRITIS OF TYPHOID FEVER

Typhoid fever may be complicated by involvement of various joints, including the spine. In the latter case, the lower spine is most frequently affected and the condition is generally characterized by marked lifting of bone with a tendency to bridging of the vertebrae through fusion of vertebral spurs.

TUBERCULOUS RHEUMATISM

Aside from the destructive type of tuberculous arthritis already discussed a form of tuberculous rheumatism resembling ordinary atrophic arthritis or rheumatic fever has been thought to exist. In 1900, Poncet suggested the possibility of the existence of such an entity. His views have been reiterated by a few other observers. Nevertheless, the evidence in favor of the existence of tuberculous rheumatism is, at present, far from convincing.

OTHER FORMS OF SPECIFIC INFECTIOUS ARTHRITIS

Many other specific infectious diseases may have arthritic manifestations associated with them. It would be useless to enumerate all of the specific infectious processes which may cause arthritis. Suffice it to say that any arthritic process associated with manifestations of an acute systemic infection should arouse suspicion of a relationship between the two.

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CHAPTER XXVI

NOTES ON SOME FORMS OF NONINFECTIOUS SPECIFIC ARTHRITIS

TRAUMATIC ARTHRITIS

Joints, like other tissues, may react differently to injuries of identical severity. The damage from trauma is undoubtedly dependent not only upon the type and severity of the injury, but also upon the inherent susceptibility or resistance of the joint tissues affected. For this reason a given injury may result in only temporary damage, quickly and completely repaired in one case, or severe, persistent, and even progressive degenerative changes in another. Because of its morphologic makeup and poor nutritive supply, articular cartilage is extremely vulnerable to the effects of any type of irritant, particularly trauma. If circumstances are not propitious and if adequate treatment, with rest and other measures, is not instituted early and maintained until the damage has been repaired, degeneration of the cartilage ensues, progressing in time to a hypertrophic type of arthritis.

Frankly traumatic types of arthritis are generally related to various types of injuries, such as falls, accidental blows and the like. As already stated, such exogenous trauma may initiate or add to an already existing arthritis, either of the hypertrophic or atrophic type, aggravating the basic condition.

Evidence of joint damage may become manifest within a few days after the injury. Or the acute effects of the original trauma may be slight, in which case symptoms may not be apparent immediately. The possibility of damage, however, is not excluded. Symptoms may appear weeks or months afterward, as manifestations of chronic hypertrophic arthritis.

The relation of the trauma in question to the existing symptoms must be weighed carefully before a diagnosis of traumatic arthritis is made. To arrive at accurate conclusions, the history, together with the evidence obtained on physical examination and roentgenographic study, must all be interpreted with care, on the basis of clinical experience.

The exclusion of atrophic arthritis is generally not difficult on the basis of clinical facts alone. The latter should be supplemented, however, by laboratory studies, including particularly the rate of sedimentation of erythro-

cates this is normal in traumatic arthritis and generally accelerated in active stages of atrophic arthritis

The synovial fluid in traumatic arthritis reveals a total cell count varying between 500 and 5 000 cells. The proportion of polymorphonuclear leucocytes may reach 75 to 80 per cent in acute stages but is much lower at later periods. If the injury has been severe and extravasation of blood has occurred the fluid may be either grossly bloody or yellowish in color.

The treatment of this type of arthritis is relatively simple but most important. Rest and avoidance of additional trauma even of a physiologic type is most essential.

Large effusions should be aspirated promptly. Removal of the fluid provides relief from pain and prevents distention of the joint capsule in addition to affording opportunity for its diagnostic study. Aspiration may be especially useful if hemarthrosis exists; the tendency for the formation of intra articular adhesions is thereby decreased.

Local periarticular injection of procaine solutions (Steinbrocker 1939) is sometimes indicated to relieve pain and to favor earlier use of the joint.

Physiotherapy by augmenting circulatory flow may be a distinct aid to recovery. Treatment must be continued as long as clinical evidence of reparable damage remains.

HEMOPHILIC ARTHRITIS

Hemophilia is not confined to any one organ; it may manifest itself in the joints as elsewhere. As a result of even minor injuries a patient with hemophilia may develop marked acute hemarthrosis with swelling and a variable amount of pain depending upon the degree of tension within the joint cavity. Signs of an inflammatory process—fever, local heat and redness—are generally absent although superficial ecchymoses suggesting the possible etiology sometimes appear. When such hemorrhages occur spontaneously and without a history of specific injury the possibility of hemophilia is obviously well founded.

As a result of repeated hemorrhages and the irritative chemical synovitis induced by them hyperplasia of the synovial membrane may occur. The subsynovial tissues infiltrated with blood may thicken markedly. The edematous and hyperplastic synovial membrane may be restored to normal following absorption of the hemorrhagic exudate after single acute hemorrhages but there is nevertheless a tendency for the synovial hyperplasia to become permanent after such repeated injuries. The hyperplastic synovial pannus may erode the articular cartilage through pressure secondary hypertrophic arthritic changes appearing in time. Roentgenographically the

shadow cast by the bloody effusion of acute hemophilic arthritis is much more dense than that cast by serous effusions in the usual types of synovitis. When hemorrhage occurs into the capsular structures also the blood pigment may cast a shadow which may be mistaken for periarticular calcification.

The diagnosis of hemophilic arthritis depends largely upon recognition of the fact that hemophilia exists. The development of abrupt swelling of joints in a young man either spontaneously or after relatively trivial injury should stimulate further questioning as to the possible existence of a bleeding tendency and as to the occurrence of similar transitory episodes of arthritis in the past. Study of the blood should then be carried out to confirm the diagnosis of hemophilia.

Treatment. For the acute episode complete rest and splinting of the joints either by means of compression bandages or splints should be provided. It is obvious that incision and even aspiration must be avoided if a possibly fatal hemorrhage is not to be incurred. When the probability of hemophilic arthritis exists and yet aspiration seems indicated for diagnostic purposes it should be carried out with a small needle in order to minimize the danger of further bleeding. Further systemic treatment of the hemophiliac should also be carried out but it is usually not very successful.

ALLERGIC ARTHRITIS (ARTHRITIS OF SERUM SICKNESS)

It is logical to assume that joint structures are not immune to offending *allergens in susceptible individuals*. The production of *intermittent hydrarthrosis* by food allergy has been fairly well established at least in a few cases but definite etiologic relationship between food or other allergens and a specific form of joint disease has not yet been satisfactorily described. It is possible that given allergic factors in atopically sensitive individuals may aggravate existing chronic arthritis regardless of its type but again proof of such a relationship has been difficult to establish.

The acute polyarthritis that may develop in the course of serum sickness is really the only true type of allergic arthritis known at present. It generally develops within from seven to twelve days after the administration of certain therapeutic or prophylactic sera and is accompanied by the usual constitutional manifestations of serum disease. *Individuals vary in their susceptibility to serum reactions in some serum sickness and its accompanying arthritis may not appear for as long as three weeks after the patient has received an injection of serum in others who are more susceptible or who have previously received an injection of serum an accelerated serum*

reaction may appear even on the second or third day after the injection. Aside from such differences, however, the incidence and severity of serum disease and arthritis are directly proportionate to the amount of serum administered. Of all the sera employed, horse serum is most likely to produce serum reactions.

Serum sickness is generally ushered in with fever and an urticarial, intensely itchy rash. Sometimes it is of an erythematous or maculopapular variety and not urticarial. Enlargement of lymph nodes and either local edema at the point of injection or edema of the face may develop. Fever and malaise generally occur, the degree depending upon the severity of the serum reaction. During the attack, leucopenia generally appears, it may later be followed by an increase in the number of eosinophils. Albuminuria from renal irritation is not uncommon, it disappears when the serum sickness abates.

In approximately 20 per cent of patients who develop serum disease, marked symptoms of joint involvement may appear within from two to five days after the onset of serum sickness. Milder forms of arthritic symptoms occur in another 30 per cent of such patients. The wrists, knees, ankles, the small joints of the hands and feet, and the elbows are most commonly affected although any joint may be involved. In milder cases there is merely joint pain and stiffness which disappear spontaneously after several days. In more severe cases, however, the joints may be red, hot, swollen, and quite painful, the process resembling closely an attack of acute rheumatic fever. Such severe articular manifestations of serum sickness may persist for from five days to a week, and even for several weeks. Eventually, however, all arthritic manifestations disappear, without residual damage. The joint fluid is turbid from the presence of leucocytes; these may number as many as 20,000 cells per cubic millimeter. A precipitin test may reveal the presence of the type of serum injected.

Treatment. When the arthritic manifestations are mild they may be disregarded for they will clear up spontaneously within several days. The treatment of the more severe cases has in the past been largely symptomatic. Administration of salicylates and application of heat generally afford relief from pain. Repeated injections of epinephrin (1 to 1,000) in doses of 0.5 to 1 cc. may relieve the itching, as does calamine lotion with 2 per cent phenol. Foshay and Hagebusch have recently reported that the administration of histaminase, either orally or intramuscularly, brings marked relief in the vast majority of patients, even in those suffering from severe forms of serum sickness. They suggest the possibility that the prophylactic use of histaminase may prevent the occurrence of serum sickness or ameliorate its severity.

NEUROPATHIC (CHARCOT) JOINT DISEASE

Neuropathic joint disease is a form of hypertrophic arthritis in which disintegration of the joint is facilitated by loss of its sensibility to pain or other proprioceptive stimuli. The conditions usually responsible for such neuropathies are tabes dorsalis and syringomyelia, although other lesions of the spinal cord such as injuries which produce permanent damage to certain nerve pathways may also be responsible. To be sure the neurologic lesion is basically related to the production of neuropathic arthritis. But the ultimate mechanism whereby the hypertrophic arthritic changes are produced is really trauma from single or multiple injuries inflicted upon such joints because their usual protective mechanism has been lost. Trophic disturbances play an indirect part by inducing relaxation of the joint capsule ligaments and tendons, permitting the joints to become unstable and therefore still more vulnerable to repeated injury.

As we have already stated the pathologic changes are essentially those of hypertrophic arthritis generally of a very marked degree. The cartilage degenerates, extreme degrees of hypertrophic marginal bony proliferation occur and enormous osteophytes develop. Some of these may break off to form loose joint bodies which still further interfere with proper functioning of the joints and augment the tendency to further degeneration and hypertrophic bony change. Marginal fractures through the joint ends of the bone frequently develop. As the joint capsule and ligaments become relaxed the joints become unstable. Effusions of fluid frequently appear. The synovia may become thickened but it never proliferates into a pannus. Ankylosis therefore does not occur.

The clinical manifestations of an advanced case are so characteristic that the diagnosis is easily made, provided one is acquainted with the clinical picture. In early cases the findings may resemble those of ordinary atrophic arthritis. However the development of enlarged joints with effusion of fluid associated with slight instability on walking should arouse suspicion of neuropathic arthritis, particularly if pain is absent or negligible.

Sometimes the patient describes a rather abrupt onset of swelling causing slight discomfort but never severe pain. He is not likely to be seriously incapacitated by his joint disease. Although there is slow yet progressive difficulty caused by weakness or unsteadiness of the extremity, he is able to continue with his usual occupation. Significantly the examination reveals absence of muscle spasm despite considerable swelling. The joint may in fact show an excessive range of mobility if laxity of the capsule has already developed. Tenderness is absent. Altogether the subjective symptoms are very slight in contrast to the marked degree of change objectively

noted In tabes dorsalis, the knees are most commonly affected, the hip next, and the ankle and foot next In syringomyelia, the upper extremities and spine may be affected, as well as the legs When neuropathic arthritis is suspected, the diagnosis may be readily confirmed by examination of the pupils and deep reflexes (practically invariably impaired in tabes dorsalis) and by sensory examination, revealing the dissociation of sensation so characteristic of syringomyelia It should be emphasized that the blood Wassermann test is frequently negative in patients with Charcot joints, either because of previous treatment, or because of spontaneous reversal of the Wassermann reaction to normal in this late stage of syphilis

The cause of tabetic arthropathy is not an active syphilitic infection but rather the end result of previous syphilis of the central nervous system This distinction is important, for, once the tabetic arthropathy has developed, antisyphilitic treatment is of no avail Such treatment applied sufficiently early, before central nervous system lues has had a chance to develop, is of course, of greatest importance in the prevention of such an arthropathy Once it has developed, all that can be done is merely to prevent further damage to these joints Immobilization by means of appropriate braces provides stability and thereby minimizes further abnormal friction and traumatic insults Large effusions should be aspirated as often as is necessary for the prevention of overdilatation of the joint capsule In younger subjects, operative fusion may be desirable Effective arthrodesis, however, is not always secured in these cases

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PART VI

NOTES ON SOME MISCELLANEOUS RHEUMATIC CONDITIONS

FIBROSITIS

DUPUYTREN'S CONTRACTURE

MYOSITIS

TENOSYNOVITIS

GANGLION

SNAPPING OR TRIGGER FINGER

INTERMITTENT HYDRARTHROSIS

BURSITIS

SYNOVITIS

ARTHRITIS OF TEMPOROMANDIBULAR JOINT

MIXED FORMS OF ARTHRITIS

ARTHRALGIA

CHAPTER XXVII

NOTES ON SOME MISCELLANEOUS RHEUMATIC CONDITIONS

FIBROSITIS

Chronic fibrositis is undoubtedly the most common cause of rheumatic symptoms. In view of the frequency with which it is encountered and the distress it may cause, too little attention has been devoted to clinical, and more fundamental studies of this disease.

Chronic fibrositis is essentially an inflammatory process affecting fibrous structures anywhere but chiefly those of muscles (intramuscular fibrositis), or tendons (tendinous fibrositis or peritendinitis), or the capsule of joints (periarticular fibrositis). When the fibrositis involves the subcutaneous areolar or adipose tissue it is spoken of as "panniculitis." Still other terms, the most common of which will be mentioned, have been employed to designate the anatomic structure affected. Many authors speak of "myositis" as synonymous with fibrositis. Although it is true that muscles are affected, they are implicated only through invasion or displacement of muscular parenchyma by pathologic changes in the interstitial fibrous tissue. "Muscular rheumatism," "myalgia," and "myofascitis" are still other terms frequently employed to designate muscular fibrositis. Such involvement of muscles and periarticular structure is, of course, a part of atrophic arthritis. But fibrositis may occur independently of this condition, in this sense we speak of it now.

Fibrositis may cause much distress, but rarely does it produce serious crippling. Although the condition may be troublesome for years and refractory to treatment, it is fundamentally a benign disease. Special care must therefore be exercised in differentiating between fibrositis and arthritis, lest the patient be burdened by a false prognosis and become unduly apprehensive about the danger of crippling.

The manifestations are pain, aching, stiffness, or soreness, which may be confined to a small area or be more widely distributed. Intermittent subjective stiffness, as if the "muscles jelled" during inactivity, is the most characteristic complaint. In muscular fibrositis the symptoms are referred

to areas between the joints. The pain is not severe but of a dull gnawing or boring character. There may be slowing of movement by stiffness, but there is no limitation of the range of motion.

The stiffness is likely to be most pronounced on awakening in the morning or after inactivity. Stiffness is relieved on limbering up by exercise. Tenderness is not consistently present. Muscle atrophy does not occur except in the most severe and malignant forms of the disease. The symptoms are characteristically variable in severity, duration and situation. Intervals of freedom from discomfort are the rule. Cold, damp weather and inactivity generally intensify the discomfort; warm weather or heat frequently gives relief. In hot weather the change from the outdoor atmosphere to that of an air conditioned room may precipitate stiffness and soreness. In some cases fatigue from overuse of the affected extremity, precipitates or intensifies the symptoms.

General fatigue is another frequent accompaniment and I am convinced that it plays a most important part in producing the condition and in aggravating the severity of the distress. So often these patients complain and show evidence of nervous irritability. When the condition affects the capsule of the joint and surrounding ligaments there may be aching, subjective stiffness and soreness on movement in given directions. The patient is frequently convinced that he has arthritis, fear of its menacing potentialities may bring him to the doctor. On examination there is usually nothing however to indicate the existence of joint involvement. roentgenograms are normal as are the other laboratory findings. Tenderness areas of thickening or discrete nodules are occasionally palpable in some of the more superficially situated muscles.

Intercostal fibrositis resulting from involvement of the chest muscles is sometimes referred to as pleurodynia. Because the pain may be increased by breathing or coughing the condition is sometimes mistaken for pleurisy. Although tenderness over the chest muscles may be elicited and the respiratory excursions may be somewhat restricted a friction rub is not audible. Angina pectoris may be suspected if the discomfort is confined to the left pectoral or parasternal regions. But it is usually not difficult to rule out angina through close analysis of the history and physical findings.

We have already stated (page 274) that the indurative type of headache so often noted in cervical hypertrophic arthritis is really more likely to be a manifestation of the associated fibrositis than of the arthritis itself.

When panniculitis exists the skin is found to be adherent to the underlying structures and it exhibits some loss of its normal elasticity.

The etiology of fibrositis is not entirely clear. Some cases appear to be infectious others probably degenerative in character perhaps the result of inherently defective local circulation. In some patients the symptoms

are so closely related to and fluctuate so synchronously with weather conditions as to create the impression of a transitory abnormality in the metabolism of muscle as the cause of the condition. In some instances trauma alone or in conjunction with degenerative changes seems to be the real cause. In the aged fibrositis is probably induced by senile changes attributable to arteriosclerotic vascular lesions. Clinical observations indicate that such changes may be markedly accelerated by trauma, intercurrent infections and exhaustion states.

The pathology of this condition has not as yet been well clarified. In most cases muscle tissue is merely replaced by proliferating or more dense fibrous tissue. In others there is evidence of an inflammatory reaction either of a patchy or a more diffuse character. In such areas there is vascular congestion, perhaps edema of tissue and infiltration of leucocytes. These areas may be sharply circumscribed, constituting essentially solitary fibrositic nodules which correspond to the tender nodules detectable at times on clinical examination.

If marked degeneration of tissue occurs the area may become infiltrated with lime salts, eventually developing even typical osteoid tissue. Such changes are not indicative of any specific etiologic factor for calcification is the usual sequel to degeneration of old dense fibrous tissue. Apparently for this reason calcification is most likely to develop in fibrositis of tendinous structures producing the so-called *peritendinitis calcarea*. The next likely site for calcareous infiltration is that of muscular fibrositis.

Periarticular fibrositis (periarthritis) of the shoulder to be described (page 388) is a relatively common condition and one which may induce considerable disability if neglected. Wry neck and lumbago are two other common fibrositic manifestations.

A rare type of chronic generalized myositis occurs in which there is relentless progression of the disease until most of the muscles affected are converted into tough unyielding fibrous tissue. This condition generally described as *generalized myositis fibrosa* has been discussed by Burton (1923), Schwab (1932), Ornstein (1935) and Somers (1939). One such patient whom we observed recently presented not only a fairly advanced stage of the disease but was also found to have signs of diffuse aplasia of the bone marrow with agranulocytosis, severe anemia and purpura suggesting the possibility of fibrous replacement of bone marrow parenchyma as well as muscle.

Treatment

Treatment of fibrositis should include a comprehensive program directed toward improvement of the individual's general physical state. Many of these patients are badly in need of prolonged rest. They will gain the most

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Treatment

Treatment of fibrositis should include a comprehensive program directed toward improvement of the individual's general physical state. Many of these patients are badly in need of prolonged rest. They will gain the most

from rest instituted away from familiar surroundings, where they are also free from mental and emotional disturbances. In many cases the preliminary rest period should be supplemented by exercise, but specific traumatic factors which may be related should be avoided. Secondary anemia, hypothyroidism, or other systemic abnormalities present, should be corrected. Reassurance regarding the benign character of the trouble and elimination of the fear of deformity are conducive to mental peace and contribute to recovery.

Obvious focal infection should be removed to improve the patient's general physical state even though such foci may have no specific relationship to the fibrositis. We know of no dietary modification that is of value, we prescribe a general diet suited to the requirements of the individual patient. Gastro-intestinal abnormalities—functional or organic—should be corrected, but we have not been able to find that "intestinal toxemia" plays any specific role. We therefore deprecate the use of colonic irrigations or other intestinal alteratives which focus the patient's attention on his bowels. The ordinary analgesics may be called for to relieve pain.

The advent of air-cooling in many public places has not been a boon, but a source of distress to the patient suffering from fibrositis. These patients must avoid chilling particularly the abrupt change from atmospheric conditions of the outdoors to those in an 'air-cooled' room. Since many patients suffering from fibrositis are adversely affected by inclement or abruptly fluctuating weather conditions, a change to an equable, warm climate is sometimes advisable. Improvement attained under such conditions is not, however, likely to be permanent, symptoms recur when the patient returns to less favorable climatic surroundings. But if he is entirely free of symptoms while in a warm climate and cannot obtain relief elsewhere, a permanent change of residence may then be a way out of an otherwise disabling situation.

Physical therapy, including the application of heat in various forms, and massage, is especially useful in the management of all forms of fibrositis. Hydrotherapy is one of the most valuable measures. The 'cures of arthritis,' achieved during sojourns in spas, and for which these resorts are famous, are largely attributable to the expert employment of hydrotherapy in cases of fibrositis. In addition to such physical therapy, there is usually the effect of favorable climatic conditions, and the opportunity afforded for rest and separation of the patient from his every day problems.

Massage, following the application of any form of heat or hydrotherapy, is another important adjunct of management. The patient with chronic fibrositis tolerates more vigorous, deeper massage than does the patient with arthritis. Deep massage with the aim of breaking up fibrositic nodules

may bring about lasting relief, if the procedure is carried out expertly by those with experience

Kahlmeter (1937) reported that x-ray therapy was beneficial Sandstrom of Stockholm indicated that he employed roentgenotherapy in a large number of cases of peritendinitis calcarea in various areas, chiefly around the shoulder The therapeutic gains he reported are impressive In many cases, resorption of the lime deposits was effected It must be remembered, however, that many of these respond equally well to various more simple types of physical therapy, including infra red radiation and diathermy Moreover, spontaneous resorption of calcium deposits also occurs at times These facts indicate that although roentgenotherapy may be useful other simpler, more readily available means should not be discarded, but should, in fact, be given the first trial

Sandstrom described the details of his technique of roentgenotherapy for peritendinitis as follows

In acute cases Dose of 75 100 r every other day or every third day, till the acute symptoms have subsided Thereafter, if slight symptoms persist, treatment is continued as in chronic cases

In chronic cases Series are given of three treatments of 100 r each Between the first two or three series there is an interval of three to four weeks, thereafter the intervals are two or three months

In both acute and chronic cases the fields are varied The technique employed has been 200 kv, 6 ma, 40 cm distance, 0.5 mm Cu and 1 mm Al filter

Although perseverance in the application of the therapeutic measures described may be rewarded by relief from discomfort, chronic fibrositis is frequently subject to recurrence despite the most expert management

DUPUYTREN'S CONTRACTURE

Dupuytren's contracture is essentially a fibrositis affecting the palmar fascia The cause of this condition is not known Pathologically, there is an inflammatory process within the palmar fascia and its digital extensions, eventually culminating in marked fibrosis of the palmar connective tissue Involvement of the digital prolongations of the palmar fascia and their fibrosis produces firm, cordlike bands along the flexor surface of the fingers When an attempt is made to extend the fingers, these fibrous cords become so prominent as to create the impression of contracted flexor digital tendons As a matter of fact, the tendons generally escape direct involvement

The symptoms usually begin with a sensation of tightness in the flexor aspects of the fingers or in the palm of the hand There may be "neuralgic" pains, sometimes a sensation of burning or tingling With the contraction

of the fascia it becomes impossible to extend the affected fingers fully. As the process advances the fingers may become fixed in varying degrees of flexion. Such deformity is apt to be progressive. In addition to the cordlike contracture of the fascia one or several nodules may be palpable. Eventually the skin over the palm and flexor surfaces of the fingers becomes adherent to the underlying hypertrophied fascia.

The condition generally begins with involvement of one or two digits in one hand, later spreading to other or all fingers of that hand. Months or years later the same condition may affect the other hand. Although the cause of this condition is unknown, trauma apparently plays some predisposing part for this condition occurs more commonly among those who use their hands extensively in their occupations. Carpenters seem especially susceptible.

Treatment

Conservative treatment of Dupuytren's contracture is unsatisfactory. The only successful treatment available at present is excision of the involved palmar fascia with its digital extensions. Meyerding (1936) reported a study of this condition in 273 patients. He described the surgical procedure of excision of the contracted fascia in considerable detail and reported excellent results from this operation in some cases and less satisfactory results in others. This operation is not as simple as it may seem. Injury to blood vessels, nerves, and tendons must be avoided. Because the skin may be intimately attached to the palmar fascia, excision may lead to loss of considerable sections of skin. Skin grafting may therefore become necessary to cover defects resulting from operation.

MYOSITIS

True myositis as a primary rheumatic affection is rare. Myositis induced by infestation with trichinae or that secondary to other inflammatory processes—pyogenic or otherwise—is occasionally the cause of muscular pains. Most cases of so-called muscular rheumatism, however, are not primary diseases of muscle but intramuscular fibrositis.

TENOSYNOVITIS

Tenosynovitis, an inflammatory process of tendon sheaths, may occur as the result of trauma or infection. The tendon sheaths over the extensor surfaces of the wrist are especially susceptible to traumatic types of synovitis. The diagnosis is evident by the presence of an elongated, sausage-shaped, fluctuant swelling directly over the tendon. Ordinarily, low grade chronic

tenosynovitis produces no symptoms whatsoever, except for the evident swelling. When the inflammatory process is associated with exudation of fibrin, or if adhesions have formed within the sheaths there may be slight pain on use of the related tendons, and sometimes a slight degree of local tenderness.

In some instances of acute tenosynovitis, gout is the etiologic factor, in others, the condition is gonococcal in origin. Such specific types of tenosynovitis require measures of treatment applicable to the specific disease causing them, other types frequently respond simply to rest. If the swelling is persistent or if adhesions limit motion of related tendons incision of the sheath may be required.

GANGLION

A ganglion is essentially a localized tenosynovitis or a cystic extension of synovia from a tendon sheath or adjacent synovial sac. It appears most often on the dorsal aspect of the wrist and may persist for years without causing symptoms, except for the presence of the projecting fluctuant mass. Treatment consists of excision of the ganglion, or of subcutaneous rupture of the cyst by sharp pressure over the mass with the thumb, while the wrist is acutely flexed. Aspiration of the ganglion and obliteration of the sac by means of sodium morrhuate has been recommended. Since the ganglion may represent herniation of synovia from an adjacent joint, however, such a procedure is not devoid of the danger of inducing arthritis by the injection.

SNAPPING OR TRIGGER FINGER

A nodular thickening of a tendon, apparently induced by trauma, and occurring generally in the region of the metacarpophalangeal joint of the middle finger, may produce the so called snapping or trigger finger. The name is derived from the snapping sensation experienced when the thickened part of the tendon slips past a narrowed tendon sheath.

Splinting the finger in the extended position for a period of from four to six weeks may effect a cure. When such conservative management fails, surgical incision with removal of the nodule and excision of the constricting portion of the tendon sheath must be carried out.

INTERMITTENT HYDRARTHIROSIS

During the course of atrophic arthritis, effusion of synovial fluid may occur, particularly in the knees. The hydrops may vary in degree from time to time, or may exhibit a tendency to periodic recurrence after intervals of

relative freedom, this continuing until the synovial membrane becomes permanently thickened and villus formation occurs. Subsequently the effusion is likely to become permanent. This process is essentially one phase of atrophic arthritis, the hydrops being merely symptomatic of the underlying condition.

True intermittent hydrarthrosis (sometimes described as idiopathic hydrarthrosis) is apparently a different entity. To the observer it presents certain fascinating, if discouraging, peculiarities, for the etiology of the condition is frequently obscure.

There is essentially a spontaneous occurrence of hydrops in one or more joints generally in the knees, the effusion remaining for several days or a week or more being gradually absorbed, until the knee recovers its perfectly normal functional integrity. After a variable interval of complete freedom, the condition recurs, and may follow a chronologic cycle identical with the first attack. In many cases the cycles are repeated with clockwork regularity, sometimes for years. In most cases, however, the duration of the hydrops and the intervals between different attacks vary in duration.

A feeling of tightness and stiffness caused by distention of the joint is usually the only symptom, pain is, for the most part, absent. The general condition of the patient is usually good. Between attacks there are no indications of abnormality in the affected joints and the roentgenograms appear normal. The synovial fluid usually contains an excessive amount of albumin and an increased number of leucocytes.

The etiology of the condition is generally obscure. In some cases trauma appears to be a precipitating cause, in most others no responsible antecedent factor can be elicited. Some have been traced to infection with *Brucella abortus*, others may be of allergic origin (Berger, 1939), still others are presumed to be caused by endocrine disturbances. The writer is inclined to believe that most cases represent an angioneurosis, analogous to Quincke's edema. The latter view has received some indirect confirmation from the observation of Weismann Netter who has reported a favorable therapeutic response in intermittent hydrarthrosis following the subcutaneous injection of ergotamine tartrate (Gynergin).

This condition may be discouragingly rebellious to treatment. The removal of foci of infection, often practiced, is likely to be disappointing. Relief from trauma by rest, bandaging, or splinting may be useful. Local physiotherapy is generally of no avail, but hyperthermia has been reported of value in a case attributed to brucellosis. Repeated aspiration of the joints has been recommended. The report of successful interruption of the process with ergotamine tartrate encourages further trial of that therapeutic agent. The elimination of offending allergens from the diet apparently brought about cure of the recurring joint effusion in the case of intermittent

hydrarthrosis, of allergic origin, reported by Berger (1939) As a last resort, synovectomy has been practiced, with apparently successful termination of the process

BURSITIS

Bursae, sacs lined by endothelium, are situated over joints, or between bony prominences and overlying muscles, tendons or skin Certain bursae, such as the subacromial, prepatellar, and olecranon, are present normally, *others develop within connective tissue through long continued trauma*, such as may be induced by pressure or friction Examples of such adventitious types of bursa are those which develop over a calcaneal spur on the heel, or over the head of the metatarsophalangeal joint of the great toe when a hallux valgus exists

Resembling in their anatomic structure the synovial membrane of joints bursae are subject to the same affections as the synovia Bursitis may be induced by trauma or infections of various sorts Bursitis is not infrequent in gout, the olecranon bursa being especially liable to involvement

The pathologic reaction is generally manifested by an outpouring of fluid which, in infectious types of bursitis, may become fibrinous or frankly purulent *As a result of organization of the inflammatory exudate in more chronic forms of bursitis*, thickening of the wall of the bursa by proliferation of endothelial cells and adhesive changes may develop Calcium is sometimes deposited within the wall of the sac

The severity of the symptoms varies with the acuteness of the process and the underlying cause In low grade, irritative processes resulting from trauma there may be only slight soreness directly over the bursa and perhaps pain on motion of the overlying muscle or tendon In more acute cases the bursa may become distended with fluid and exquisite tenderness and pain may develop Superficially situated bursae may become palpably thickened

In chronic traumatic bursitis treatment includes protection of the bursa *from trauma* This may be accomplished by the use of bandages or pads which eliminate pressure Dry heat, or that applied by means of hot compresses, may allay inflammation and relieve discomfort Salicylates usually suffice to control pain, in acute cases morphine may be required Aspiration of free fluid may be of temporary value, but the fluid is likely to reaccumulate unless the causative factor has been eliminated at the same time In frankly suppurative types of bursitis early incision and drainage is advisable Obliteration of the sac by sodium morrhuate, after aspiration of the fluid, has been recommended A superficially situated bursa, which is the seat of a chronic inflammatory process, may be readily excised Such a procedure

is especially applicable in olecranon bursitis or in the management of painful bursae associated with bunions in which case the bursa may be excised during operative correction of the hallux valgus

Various occupational types of bursitis occur the radiohumeral bursitis (tennis elbow) of the tennis player, the prepatellar bursitis of the house maid or nun, and the olecranon bursitis of the miner or draftsman

The pain associated with calcaneal spurs is really the result of calcaneal bursitis, as can be readily proved by the relief from pain which follows treatment, either with physiotherapy or obliteration of the bursa, even when the bony spur is otherwise disregarded When the pain of calcaneal bursitis is not eliminated by conservative measures, however, the bursa as well as the overlying calcaneal spur should be excised Surgical resection of calcaneal spurs should not be attempted hastily, since conservative management frequently suffices to relieve the patient of discomfort

Subacromial (subdeltoid) bursitis is a clinical condition which is met in practice so frequently that it is discussed in more detail elsewhere (page 3-8)

SYNOVITIS

Although traumatic or infectious types of synovitis may occur without involvement of any other parts of the joint, it is usually difficult to draw the line between primary synovitis and synovitis that is merely symptomatic of an arthritis In traumatic synovitis resolution of the inflammatory process is usually effected by thorough immobilization with bandaging Aspiration of free fluid or bloody extravasations is sometimes necessary Pyogenic types of synovitis require surgical drainage Other specific forms of synovitis demand attention to the specific etiologic factors

ARTHRITIS OF TEMPOROMANDIBULAR JOINT

In severe cases of atrophic arthritis especially when the condition is wide spread, the temporomandibular joints may be affected in the same way as other articulations Pain and varying degrees of limitation of full motion are the results observed in the jaw Such symptoms may also occur unilaterally in individuals who are entirely free of arthritic manifestations elsewhere A tendency to locking of the jaw is frequently noted Many of these cases are apparently initiated by trauma with injury to the cartilage and secondary hypertrophic arthritic changes When advanced, the joint may become badly disorganized, as is evident in roentgenograms which reveal marked irregularity in the joint outline caused by hypertrophic spurs

Costen emphasized an important group of symptoms, which include headache and pain referable to the ear, associated with disturbed function of the temporomandibular joint. Many of these symptoms are caused by dental malocclusion and they disappear when the malocclusion is corrected.

When limitation of full action of the temporomandibular joint exists manipulation under anesthesia has on occasion proved successful in increasing the range of function. In atrophic arthritis, such local therapy supplements systemic measures that are required. Diathermy is of some value *in the after treatment*. When the joint is badly disorganized, arthroplasty or even better, excision of the condyle may be performed.

MIXED FORMS OF ARTHRITIS

Just as syphilitic aortitis may be engrafted upon a degenerative, atheromatous aorta, so atrophic arthritis may be superimposed upon a pre-existing degenerative type of joint disease. Or a specific infectious type of arthritis may develop in a patient previously subject to either a degenerative or atrophic type of arthritis. Such mixed types of arthritis present, of course manifestations of both existing joint disorders. When the symptoms or physical findings do not run true to any one type, the possibility of such mixed forms should be considered. The treatment must take into consideration the influences underlying both forms of joint disease.

ARTHRALGIA

Some rheumatic pains cannot be attributed either to joint or fibrositic changes. They represent physiologic effects of a large variety of systemic disturbances, not all of which are clinically discernible. Some of the conditions known to produce fleeting or more persistent, arthralgias are the menopause, secondary anemia, hypothyroidism, diabetes, perhaps gout, and nervous exhaustion. In some cases the pain represents an escape mechanism for a chronic psychoneurotic anxiety state. This condition has been referred to as psychalgia.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527]

Myositis

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PART VII

PAIN IN THE SHOULDER AND ARM

SUBACROMIAL BURSITIS

PERIARTHRITIS OF THE SHOULDER

CERVICAL RIB AND THE SCALENUS ANTICUS SYNDROME

NERVE INVOLVEMENT AS A CAUSE OF PAIN IN THE CERVICAL SPINE,
SHOULDER, AND ARM

CHAPTER XXVIII

SUBACROMIAL BURSITIS

One of the most specific and most easily diagnosed entities in relation to shoulder pain is subacromial (subdeltoid) bursitis

In 1904, Dr E A Codman of Boston pointed out the clinical relation ship between subacromial bursitis and the painful shoulder Since that time the pathology of this condition, its clinical manifestations, and its treatment, have been studied intensively and reported in a long series of publications by Codman and others In 1934, Codman summarized the entire subject in his book, "The Shoulder," one of the classics of American medical literature This book, dealing not only with a discussion of every phase of subacromial bursitis, but also with many other closely related entities, should be consulted by anyone seriously interested in this subject

ANATOMICAL RELATIONSHIPS

For clinical purposes, the subdeltoid bursa may be said to have its center over the upper portion of the greater tuberosity of the humerus (Fig 105) When the arm is abducted, the bursa slides with this portion of the humerus under the protective shelf of the acromion With the arm at the side, two-thirds of the bursa is beyond the acromion and subdeltoid in situation In full abduction, all of it is under the acromion The insertion of the tendon of the supraspinatus muscle at the greater tuberosity of the humerus is directly under the floor of the bursa In youth this tendon is well formed and a rather substantial structure In advanced years it becomes thinned, until it may be represented by only a thin fibrous strip

ETIOLOGIC FACTORS

The subacromial bursa is subject to the same afflictions as are other bursae Trauma may produce increased effusion, nonspecific infections may attack it, purulent bursitis may develop metastatically After the third decade, a most frequent predisposing factor in the production of subacromial bursitis is degenerative change within the tendon of the supraspinatus muscle lying as we have indicated, under the floor of the bursa

Trauma of some sort appears to be the most important precipitating

factor in the etiology of subacromial bursitis. The trauma may result from a major injury, or more often from repeated minor injuries occupational or otherwise. A thinned out degenerating partially frayed supraspinatus

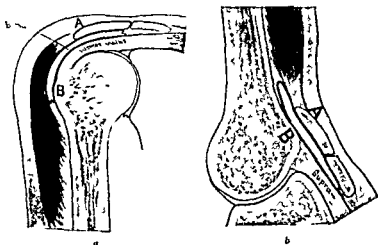


FIG. 105. Coronal sections of the shoulder showing the anatomic relations of the subacromial bursa. a The arm in anatomic position and the bursa largely subdeltoid in situation. b The arm elevated and the bursa largely under the acromion.

Note the relation of the floor of the bursa to the insertion of the supraspinatus tendon at the greater tuberosity of the humerus. (From Codman, *The Shoulder*, Thomas Todd Co., 1934.)

tendon, damaged from a series of minor injuries over a number of years, is of course more susceptible than a normal tendon to the effects of acute injury. An explosive attack of subacromial bursitis will then be attributed to the last injury, actually the groundwork has been laid long before. Actual tears of the supraspinatus tendon occur and may induce a subacromial bursitis. Accurate diagnosis of this type of accident is most important, because preservation of function in these cases, which is so essential, depends upon early suture of the tear in the supraspinatus tendon.

Focal infection by itself does not seem to be important in causing subacromial bursitis. Cultures of affected bursae by Codman and others have been consistently negative, yet it is conceivable that focal infection may localize in, and aggravate, a degenerative or traumatic lesion in the subacromial bursa, or in the subjacent supraspinatus tendon.

PATHOLOGY

As a result of extensive study, Codman concluded that most cases of subacromial bursitis originate in the tendon of the supraspinatus muscle,

lying directly under the floor of the subacromial bursa. He believed that degenerative changes and perhaps even actual small tears in the thin relatively inert supraspinatus tendon, may develop repeatedly without pain

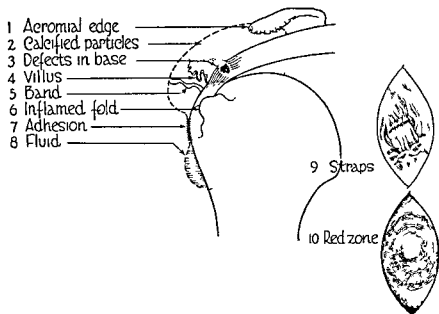


FIG. 106 Diagrammatic sketch indicating the pathologic manifestations in subacromial bursitis (From Codman *The Shoulder* Thos. Todd Co. 1934)

He suggested that the pain of subacromial bursitis appears only when the process in the tendon extends into the adjacent subacromial bursa.

The earliest pathologic change in acute subacromial bursitis consists of diffuse congestion and engorgement of blood vessels just beneath the lining cells of the bursa. This congestion is most pronounced over its floor, extending also to the underlying tendon of the supraspinatus muscle. In addition there is marked edema of and effusion of fluid into the sac which may become definitely distended. The effusion may be clear and sterile, purulent or hemorrhagic depending upon the etiologic factor.

The integrity of the lining cells of the bursa may be maintained. When the acute process is more intense, however, the inflammatory process may lead to actual necrosis of the lining endothelial cells. In that event a breakdown of small capillaries may occur with exudation of blood serum and the formation of a fibrinous exudate (Fig. 106). The inflamed area in the floor of the bursa may become infiltrated with calcium. This calcific deposit, essentially an amorphous collection of fine powdered inorganic calcium salts, may be aggregated into a single relatively compact mass or it may be scattered more diffusely over the affected area, it may be extruded into and spread diffusely throughout the bursal sac (Fig. 107).

Not only the floor of the bursa but the entire sac participates to some extent in the inflammatory process. There is even associated inflammatory change in other tissues immediately surrounding the inflamed bursa.

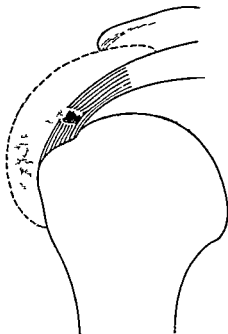


FIG. 10. Diagrammatic sketch showing a calcareous deposit within the supraspinatus tendon bursting into the subacromial bursa. (From Codman: *The Shoulder*, Thomas I. Co., 1934.)

Such an acute inflammatory process may subside in time with complete resolution of edema and congestion, thorough absorption of the fibrous exudate, and even fairly complete resorption of the calcific deposit. In other cases the entire process resolves except that a residue of calcium remains within the bursa or over its floor.

When the extent of the inflammatory change has been more pronounced the process more prolonged, and particularly if treatment has been neglected, fibrous adhesions are likely to develop between the walls of the subacromial sac. These adhesions may or may not obliterate the bursa. Even more important, perhaps, are similar adhesive changes which may develop between structures surrounding it, for such adhesions between fasciae or muscles about the shoulder may subsequently cause marked limitation of shoulder function. This in turn may lead to atrophy of muscles about the shoulder girdle, permitting still further adhesive changes and additional embarrassment of function.

CLINICAL MANIFESTATIONS

The manifestations of subacromial bursitis depend on the stage of the process at which the patient is seen, on the character of the pathologic change that exists, and on whether the attack is recent and acute or merely residual of a former attack, the acute stage of which has passed

The usual history is that of rather sudden onset of pain, most intense at the point of the shoulder, the pain may be so severe that opiates fail to relieve it. A day or two of such suffering and resulting loss of sleep produce the characteristic anguished expression. The patient with a severe, acute attack of subacromial bursitis presents a picture easily recognized. He keeps the affected arm close at his side, the elbow flexed, and the lower arm across the body, this is supported with great care by the other arm. Even at rest he is harassed by an acute, piercing pain which makes sleep impossible. He fears the slightest movement, he cringes with any attempt at abduction of the arm or palpation over the shoulder. He may perhaps be induced to elevate his arm forward, and, if his confidence can be won and he can be induced to relax completely, even a slight degree of passive motion may possibly be carried out, but, because of the violent pain and muscle spasm it produces the full range of abduction and rotation is utterly impossible.

Standing behind the patient, the observer may note a slight fullness where the bursa is situated. There is exquisite tenderness over the entire shoulder region, but especially just below the tip of the acromion and for about an inch distal to it. A low grade fever and slight leucocytosis may be found.

The patient usually localizes his pain quite accurately to the situation of the subacromial bursa but in many cases the area of pain is more diffuse. It may involve most of the shoulder or may radiate to the neck or to the region of the deltoid insertion, it may extend down the arm to the elbow, and occasionally even to the finger tips. However, even when the discomfort is widespread it is most intense directly over the bursa.

Though at first the patient may be unable to recall any unusual trauma seemingly related to his condition, he may later remember pushing or lifting a heavy object with the arm in abduction. The condition may have appeared after strenuous use of the shoulder within the past twenty-four hours, after unguarded, sudden lifting of the arm, or after a fall on the outstretched hand.

The roentgenogram of the shoulder may reveal no abnormality, but, in patients in or beyond the fourth decade, an irregularly rounded, opaque shadow is frequently seen just outside the tuberosity of the humerus. This

shadow represents the granular deposit of lime salts within the insertion of the supraspinatus tendon just beneath the floor of the bursa (Fig. 108)

With or without treatment the pain reaches a certain intensity, wanes



FIG. 108. Roentgenogram of the shoulder in subacromial bursitis showing the bursa distended by fluid and containing calcific material (From Codman: *The Shoulder* Thos. Todd Co. 1934)

and then gradually disappears leaving only a dull soreness which may remain for days or weeks. However if the patient is allowed to keep the arm at his side until all pain has disappeared adhesions are likely by that time to have developed within the bursa and between its surrounding structures. These may limit abduction of the arm after all pain has gone. In some cases no more than 15 to 20 degrees of lateral abduction is possible and external and internal rotation may be proportionately restricted. These changes have given rise to the designation "frozen shoulder" a term which is accurately descriptive. In such instances atrophy of the muscles about the shoulder girdle generally appears. The slight persistent soreness in the shoulder may be aggravated by attempts to raise the arm. Although a localized area of tenderness may still be discernible it is less sharply defined than in acute cases. A roentgenogram at this stage reveals diffuse decalcification of the upper end of the humerus in addition to the calcified mass in the supraspinatus tendon already described.

A chronic form of subacromial bursitis occurs, in which there is dull pain, sometimes very annoying, but never as severe as in acute bursitis. More sharp pain may be induced from time to time by sudden motion, particularly abduction, especially as the arm reaches an angle of 90 degrees. There is also soreness on lying on the affected side. As a rule the range of mobility is not impaired, it may be slightly restricted by pain or mechanical factors. A history of a previous acute attack with recovery from all but the chronic symptoms just described is sometimes elicited, in other cases the condition develops without any antecedent acute episode. An area of localized tenderness may be noted directly over the situation of the bursa. If such tenderness exists, it disappears as the patient abducts the arm beyond a right angle, this is because the bursa then slides under the acromion, and is no longer within reach of the palpating finger. There is never more than a slight degree of muscle atrophy over the shoulder.

In this chronic type of subacromial bursitis a roentgenogram may reveal relatively little change. Sometimes there is a linear area of increased density over the greater tuberosity of the humerus, particles, or larger masses, of calcified material in the supraspinatus tendon may or may not be visible, some generalized atrophy of disuse may be noted in the upper end of the humerus if functional impairment has existed for any length of time.

TREATMENT

Even without specific treatment, or despite desultory management, many patients with acute subacromial bursitis eventually become free of acute pain. Residual aching or soreness may persist for many months, even for years, but in many of these cases a fairly normal or even a full range of function remains. In those in which some impairment of shoulder function persists, recurrent episodes of acute bursitis are likely to occur, with more and more structural and functional impairment resulting.

Treatment of subacromial bursitis aims at relieving pain and restoring the fullest integrity of shoulder function as early as possible. Relief from pain alone is not enough, fixation of the arm in the adducted position must be prevented. Both of these objectives may be achieved most easily by the injection of novocaine into the bursa. When the pain has been severe and the patient is unduly apprehensive, the injection may have to be performed under general anesthesia. In most cases, however, preliminary infiltration of the skin and underlying structures with novocaine permits the insertion into the bursa of a 16 or 18 gauge needle through which 20 cc. or more of 2 per cent novocaine solution may then be injected in fractional amounts. The solution is reaspirated along with some of the exudate present in the

bursa An attempt is also made to pierce the floor of the bursa in order that the tendon of the supraspinatus muscle may be infiltrated with novocaine. At the same time, any calcified material embedded in the tendon may be extruded into the sac of the bursa and aspirated through the needle with the novocaine solution. Any calcific material remaining is frequently absorbed spontaneously or in the course of physiotherapy which is subsequently employed. It is thought that lacerating the inflamed floor of the bursa and extruding the calcified mass into the sac reduces tension and thus affords relief from pain. After the bursa has been washed out, the capsular tissues may also be infiltrated with novocaine solution.

In acute cases with severe pain of recent onset, Patterson and Darrach have employed through and through irrigation of the bursa with novocaine and later saline solution, employing two needles inserted into the sac.

Following such procedures marked (sometimes complete) relief from severe pain may be effected almost immediately, thus permitting abduction of the arm to a right angle. By means of an abduction (aeroplane) splint or a light plaster cast the arm is then maintained in a position of abduction and external rotation until all tenderness about the shoulder has been resolved.

The arm is not left immobilized indefinitely, however. Often within forty-eight hours, and practically always within four or five days, passive motion of the shoulder is begun. The range of passive exercise is confined within the limits of pain and is not pushed too vigorously at first. At the start no attempt is made to go through anything like the full range of shoulder motion. Exercises are generally preceded by the application of heat. After each period of exercise the arm is returned to the splint and allowed to rest in the abducted position. Within a week or two, sometimes earlier, active motion from the resting point is begun. The range of active exercise is gradually increased until restoration to normality is attained.

Simple needle puncture of an inflamed, distended bursa sometimes affords relief from pain. Weeks and Delprat found that multiple punctures, even without the injection of novocaine, and without withdrawing the fluid from the bursa, relieve tension within the sac and thus, apparently, causes the violent pain to abate.

Brachial plexus block with aqueous procaine solution has been found of value by Steinbrocker (1939). It might be useful in relieving pain sufficiently to permit abduction of the arm and splinting in that position.

Codman and others have suggested surgical exploration and drainage of the acutely inflamed bursa. Its roof is incised through a small vertical opening over the region of the tuberosity. The inflamed base of the bursa is then penetrated with the point of a knife, permitting some of the pastylike

calcified material to exude. The remainder of it is then removed by gentle curettage, any adhesions present are separated, a portion of the roof of the bursa is excised and the incision is closed. Postoperatively the arm is maintained in abduction. Physiotherapy and active exercises are begun on the second day and increased in range and frequency until normal restoration of joint function is achieved.

Roentgen irradiation has been reported to be of value (Lattman, 1936). In acute cases the symptoms may be aggravated during the first twenty-four hours following a treatment, but there is subsidence of pain and increased mobility in the next twenty-four hours. One or two such treatments are said to suffice to relieve pain completely. In some instances, calcified deposits (present before such therapy was instituted) disappeared within two months.

In cases in which the symptoms are not extremely severe, particularly in those in which the pain is not sharply circumscribed to the region of the bursa, conservative treatment with physiotherapy—heat, light massage, and active exercises—usually suffices. The heat may be derived from an infra-red or radiant heat lamp or from short wave diathermy, it does not seem to matter much which of these modalities is employed. The calcific deposits (revealed in the roentgenogram before physiotherapy is begun) are in many cases largely or entirely absorbed after weeks or months. However, relief from pain does not depend upon disappearance of the calcium salts; many patients who are entirely relieved, retain the calcium shadow intact. If the patient is free of pain it is not logical or necessary to remove such calcific deposits surgically.

In the chronic type of subacromial bursitis with adhesions, manipulation of the shoulder under general anesthesia is usually necessary in order that these adhesions be broken and shoulder motion restored. Such manipulation must be done with great care, lest a fracture of the humerus or tears in the rotator tendons of the shoulder be induced. Immediately following manipulation the arm is placed in an abduction splint and the shoulder is treated essentially as a case of acute subacromial bursitis. Heat, passive exercises, and later active motion are instituted. The after-treatment of chronic adhesive subacromial bursitis may require many weeks or months before full restoration of shoulder function is obtained.

If chronic adhesive subacromial bursitis is associated with calcification, direct surgical attack may be advisable. The adhesions are separated, the calcareous deposit is removed, and the shoulder manipulated to break up adhesions that might be present between the tendons of the short rotator muscles. Intensive physiotherapy including exercises aimed at rehabilitation of the shoulder are then instituted.

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CHAPTER XXIX

PERIARTHRITIS OF THE SHOULDER

SYNONYMS *Stiff, painful shoulder, peritendinitis*

Most cases of arthritis of the shoulder or subacromial bursitis have associated periarthritic changes, but periarthritis of the shoulder may occur as the predominant entity, which should then be differentiated from other conditions causing shoulder pain.

Periarthritis of the shoulder develops most commonly after the age of forty and more often in people who do manual rather than sedentary work. There is a greater tendency to involvement of the right shoulder than the left but the condition is often bilateral.

ETIOLOGY

The basic causes of periarthritic changes at the shoulder are probably essentially the same as those of tendinous fibrositis anywhere.

Codman and Fowler believe that trauma, causing tears or fraying of the supraspinatus tendon, is the basis of most cases of periarthritis of the shoulder. Trauma does appear to play a role in many cases. The condition may develop following direct injury resulting in dislocation of the shoulder or gross tears of the supraspinatus tendon, and a large number of patients relate a history of less obvious injury preceding the onset of symptoms. In others, the trauma may have been readily ignored or forgotten. But that trauma is not the only factor concerned, is indicated by the fact that a certain proportion of patients starting with periarthritis of the shoulder eventually develop generalized rheumatic manifestations, either fibrositic or frankly arthritic in nature. Actually, it is not uncommon for atrophic arthritis to be preceded by the development of periarthritis of one or both shoulders, a condition which may appear months or years before the advent of the generalized arthritic process.

Focal infection does not appear to be a primary etiologic factor in this condition. There is reason to suspect, however, that with the fundamental fibrositic changes established, there is increasing susceptibility to the aggravating influence of focal infection.

In an analysis of 200 cases, Dickson and Crosby concluded that focal infection and glandular dysfunction were more important than trauma as etiologic factors

It has been impossible to find consistently any specific metabolic derangement associated with periartthritis of the shoulder. However, the condition is likely to develop during middle age, frequently during the menopause, and, in our experience, commonly among older diabetics and in people who present various grades of hypothyroidism. We therefore suspect that various metabolic disturbances, even though not primarily related, may aggravate the process.

We have seen the condition appear in the wake of general physical debility, or following shortly after an attack of acute cardiac failure induced by a variety of lesions, including coronary occlusion. In these cases the condition was usually bilateral, and in some of them the periartthritis of the shoulder was merely part of a generalized fibrositic process.

PATHOLOGY

Owing to the paucity of material available for pathologic study, the changes of periartthritis of the shoulder have never been clearly ascertained. We must therefore construct the pathologic picture from data obtained from occasional postmortem examinations or in the course of operations such as have been reported, especially by Codman.

Fundamentally, the changes are those of fibrositis as seen anywhere. In this instance, however, they affect either the supraspinatus tendon alone, or a group of tendons about the shoulder. In most cases the changes appear to be essentially degenerative, not inflammatory, in character, with fragmentation, sometimes actual fraying of tendinous fibrous tissue. The process may affect many tendons along their fascial planes, adhesions may form between these, resulting in limitation of motion and the so-called "frozen shoulder." In a few there is an additional deposition of calcium salts at the site of maximum injury. The point of insertion of the supraspinatus tendon, under the floor of the subacromial bursa, is especially liable to such calcareous infiltration. As we have already indicated, this change may be followed by involvement of the adjacent subacromial bursa with the production of a bursitis.

CLINICAL MANIFESTATIONS

In many respects the manifestations of this condition resemble those of subacromial bursitis, discussed in the previous chapter (page 378). In pen

CHAPTER XXIX

PERIARTHRITIS OF THE SHOULDER

SYNOVIAL *Stiff painful shoulder, peritendinitis*

Most cases of arthritis of the shoulder or subacromial bursitis have associated periarthritic changes but periarthritis of the shoulder may occur as the predominant entity, which should then be differentiated from other conditions causing shoulder pain.

Periarthritis of the shoulder develops most commonly after the age of forty and more often in people who do manual rather than sedentary work. There is a greater tendency to involvement of the right shoulder than the left but the condition is often bilateral.

ETIOLOGY

The basic causes of periarthritic changes at the shoulder are probably essentially the same as those of tendinous fibrositis anywhere.

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CLINICAL MANIFESTATIONS

In many respects the manifestations of this condition resemble those of subacromial bursitis, discussed in the previous chapter (page 378). In per-

arthritis of the shoulder there is, however, more stiffness about the shoulder than pain and tenderness, a sensation of weakness and a tendency to tiring on use of the arm, and progressively increasing restriction of the range of motion. The tenderness and pain are neither as severe nor as sharply localized as in subacromial bursitis, it radiates over an extensive area below the tip of the acromion, into the deltoid region, or along the outside of the arm to the fingers, and it sometimes extends upward over the shoulder girdle as well.

The patient may relate that his symptoms developed within a day or two after some injury such as might be inflicted by overuse of the arm in abduction or by a sudden unguarded upward thrust of the arm.

Motion particularly raising the arm or attempting to rotate it, aggravates the pain which becomes progressively more disabling as the range of function becomes more restricted. It may be that the sharp, stabbing pains occurring in advanced cases when certain motions are attempted are caused by actual pulling on adhesions between the fascial planes.

The pain of periarthritis may become so severe as to require morphine for relief. In such cases there is possible involvement of the adjacent subacromial bursa although it may be difficult to prove this fact, because the symptoms of the more diffuse periarthritis predominate.

There is gradually increasing limitation of motion, especially abduction, internal and external rotation, forward and backward flexion is not disturbed and is painless. In this respect again the symptoms resemble those of subacromial bursitis. In milder cases abduction may be carried out without pain until the arm is passing through an arc of from 70 to 90 degrees, at which time pain appears, it disappears again as abduction is carried beyond that point as the arm is lowered, the pain recurs during the corresponding painful arc.

At first, the limitation of motion is caused by pain only and eventually by adhesions. When the latter exist the examiner gets a sensation of general tightness from contracture of periarthritic structures as he attempts to abduct or rotate the arm. If the range of mobility has been limited for some time, atrophy of the muscles about the shoulder and upper arm is usually evident.

LABORATORY DATA

Generally the blood count and sedimentation rate are normal. A number of these patients have a definitely lowered basal metabolic rate, the significance of which in relation to the periarthritis, is not entirely clear. Even when the periarthritis is associated with calcification in the supraspinatus

tendon, there is no deviation from normal in the concentration of the blood calcium or phosphorus

In most cases roentgenograms usually reveal nothing abnormal in some there is evidence of calcareous infiltration at the point of insertion of the supraspinatus tendon

TREATMENT

The treatment of periarthritis of the shoulder should include systemic management of the patient as well as local treatment of the shoulder. Since the process is essentially a fibrositis, the principles applying to the systemic management of fibrositis (already described page 363) apply equally to the patient with periarthritis. We are impressed with the results from treatment which improves the patient's general condition—the correction of anemia and of metabolic errors (hypo- or hyperthyroidism or diabetes). When the periarthritis is associated with circulatory failure resulting from myocardial insufficiency, re-establishment of normal circulatory function is an essential prerequisite to management of the local condition in the shoulder. A well balanced general diet, low or high in calories, depending upon the requirements in the given case, is prescribed. No other specific alteration of diet is required. Except in diabetes, restriction of carbohydrate does not appear to be necessary. A normal vitamin balance should be maintained, but the administration of massive doses of vitamins is not indicated.

If the process is acute and the pain severe, analgesic drugs, such as aspirin, should be allowed. The addition of codeine sulphate may be necessary for a short period to relieve the most acute pain. During the acute stage, an ice cap applied to the shoulder may afford some relief from pain, but in more chronic forms of the disease, heat is more logically indicated. Short wave diathermy or roentgenotherapy may be employed as supplementary measures.

The local treatment of the shoulder is based on the principles that apply also in the treatment of subacromial bursitis (described in the preceding chapter, page 384). It is important to bear in mind the tendency to the development of adhesions between fascial planes of the periarthral structures, and the tendency to contracture of the shoulder capsule, with resulting fixation of the arm in a position of adduction. Deliberate steps must be taken to prevent such an occurrence through the use of either an abduction (aeroplane) splint, or by continuous traction applied above and below the elbow, with the arm in abduction and external rotation. Full abduction and external rotation may be impossible when the patient

is first seen, if the deformity is caused by pain and muscle spasm. Yet, if the deformity is not too marked, its correction may be attained slowly by gradual traction.

Although passive motion should be instituted as early as possible, and the arm carried through the fullest available range of abduction and external and internal rotation, motion should not be forced too soon, nor too vigorously, lest more pain and muscle spasm be created. Gradually, active voluntary motion is instituted, while the arm is still supported by the traction apparatus.

In time the range and the amount of active exercise are increased. When the patient is able to execute the exercises through the full range of motion without inducing pain or muscle spasm, the splint or traction apparatus may be dispensed with and the patient permitted to continue with the exercises alone.

If the full range of abduction and rotation is only slightly limited and the process is not acute the full range of mobility may be restored either by traction or by gradual passive manipulation of the shoulder immediately after the application of heat or diathermy.

When the normal range of motion cannot be restored by the means just described or if the adduction deformity has been present for some time and is evidently caused by adhesions, manipulation of the shoulder, with the aim of breaking up existing adhesions, should be performed. Nitrous oxide anesthesia may be employed, sodium pentothal administered intravenously is especially suitable for this type of operation. During the manipulative procedure the arm is forced through the full range of abduction and internal and external rotation, care being taken of course, to avoid excessive trauma to the soft structures involved, or to the bones which may be fractured. Following manipulation the arm is fixed in the position of full abduction and external rotation, the position is maintained by means of an abduction splint or by traction. The follow up treatment is essentially the same as that which is carried out following manipulation of the shoulder for subacromial bursitis (page 386).

In cases in which the periarthrititis may be the result of traumatic rupture of the supraspinatus tendon, surgical exploration with repair of the rent by suturing is indicated, of course. Codman and Fowler advocate more frequent recourse to surgical exploration in such cases, believing that in many the condition is the result of rupture of fibers of the supraspinatus tendon. The consensus of opinion is, however, that radical measures should not be employed as a first recourse and the soundness of this view is substantiated in practice by the satisfactory results usually obtained with the more conservative, nonoperative measures of treatment outlined.

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[For a list of books and monographs dealing with the general aspects of chronic rheumatic disease (including considerations on the subject of the present chapter) see page 527]

CHAPTER XXX

CERVICAL RIB AND THE SCALENUS ANTICUS SYNDROME

Like sciatica" in the leg so called neuritis" of the arm is a very common complaint and one which is just beginning to be differentiated through better understanding of some mechanical abnormalities of the body Painful conditions of the arm are due not only to frank inflammatory or traumatic causes, but occasionally to anatomic deviations The presence of a super numerary or cervical rib has long been recognized as a cause of pain about the arm and neck Only recently, however, has the syndrome of scalenus muscle compression been clinically recognized as a frequent cause of disability

Attention was first called to the importance of 'a rigid scalenus anticus muscle' in the cervical rib syndrome by John B. Murphy in 1905 In relieving symptoms of cervical rib compression, Adson and Coffey found resection of the scalenus muscle to be as successful as removing the rib In 1934 Naffziger performed scalenotomy as a resource in relieving patients of shoulder pain and related symptoms, in the absence of a demonstrable extra rib He published his results in 18 cases upon which he had performed section of the scalenus muscle Within the past few years this syndrome has received much attention, the many recent reports indicating its relative frequency

PERTINENT ANATOMIC CONSIDERATIONS

The scalenus anterior (scalenus anticus) muscle lies in the neck in close contact with the great blood vessels and with the nerves of the brachial plexus It arises from the tubercles on the transverse processes of the fourth, fifth and sixth cervical vertebrae, and descends almost directly to its insertion on the upper surface and inner border of the first rib, where it lies immediately adjacent to the subclavian artery A slip of the muscle often passes behind this artery as well This important vessel, in effect, runs through a narrow triangular opening formed by the rib beneath and the tendinous portions of the scalenus muscle above Hence it is subject to squeezing between the two when the rib and muscle are abnormally approximated

The medial cord of the brachial plexus, which gives off the ulnar and median nerves, also traverses this triangular space and is similarly susceptible to abnormal pressure in this region. When the scalenus muscle acts from above, as in deep inspiration, it raises the first rib. Respiratory motions therefore aggravate a pinching effect of rib and muscle upon these structures.

When an anomalous rib is present, it is usually on one side of the neck only. Bilateral extra ribs do exist, however. A fibrous extension over the vital structures of the neck, from even the smallest, most rudimentary rib can produce severe symptoms. Sooner or later especially in people who perform manual work, the presence of the extra rib in the posterior triangle of the neck is irritating and produces symptoms. Since the structures affected—the subclavian artery and the lower part of the brachial plexus—are the same as those involved by an abnormal scalenus muscle the clinical pictures are essentially similar.

INCIDENCE

True cervical ribs which produce symptoms are rare. Although a rudimentary cervical rib is present in all embryos, it atrophies in 99 per cent of individuals. Of those that persist, only about one in ten causes any symptoms. However, the syndrome of scalenus muscle compression, which is very similar to that of cervical rib, is encountered frequently. Although the general practitioner may never see a case of cervical rib producing symptoms, he has a reasonable expectation of seeing an occasional case of the scalenus muscle compression syndrome and of treating it satisfactorily.

ETIOLOGY AND PATHOGENESIS

Although the scalenus anticus syndrome is essentially a disease of adults, its causes begin at birth. The trouble often lies dormant until provoked by one of the stresses of later life. Behind the abnormality of the scalenus muscle in adulthood lies, for instance, the abnormal development of the shoulder girdle (Todd). During the growth of an individual, the weight of the upper extremity pulls down the acromial end of the clavicle while the rectus abdominis muscles pull down the sternal end. If the pull of the arm is greater than that on the sternal end of the clavicle symptoms of compression of the brachial plexus and subclavian structures on the first rib (or extra rib) may result. Greater descent of the shoulders takes place in women and in long-necked individuals. Still another predisposing factor is the abnormal origin of the brachial plexus, which, when it is derived predominantly from the thoracic instead of cervical segments, lies lower in relation to the first rib and is, therefore, more apt to be angulated (Jones).

During later years debilitating illness may cause a greater shoulder sag; the onset of pain may come on after some long or severe sickness. Such factors, too, as spasm of the scalenus muscle or myositis may result in the formation of secondary fibrous bands, which, upon organization, constrict the plexus. Such bands, possibly due to trauma or inflammation, have occasionally been encountered at operation. Any trauma or sudden twist may activate scalenus pain, especially in individuals anatomically predisposed to it. It is significant that most cases occur in women and in the right shoulder. It has been suggested that the syndrome may be provoked in some women because they drop the right shoulder in the daily act of sweeping.

CLINICAL MANIFESTATIONS

Consideration of the anatomical facts discussed helps to elucidate the symptomatology of these conditions. When the anterior scalenus muscle or an accessory (cervical) rib causes pressure in the neck, it compresses the brachial plexus or the subclavian artery, producing symptoms referable to nerve irritation and to interference with circulation. As would be expected from the low position of the medial cord of the brachial plexus, the first symptoms are generally attributable to compression of this nerve trunk. The trunk gives off the ulnar and median nerves (C8-D1). The radial nerve comes off the posterior trunk and is infrequently involved in this syndrome and even when it is, the ulnar nerve is still more severely affected.

Pain in the upper arm, about the shoulder, and about the neck or head are predominant symptoms especially when aggravated by motion of the arm or neck. This pain may be of weeks' or years' duration. Sometimes it is on the left side and radiates in such fashion as to suggest the pain of coronary heart disease. The scalenus triangle in the neck on the affected side is generally tender to pressure in comparison with the other side.

Numbness or *hypoesthesia* of one or more fingers is practically always present. As has been indicated, it is predominantly ulnar in distribution, that is, over the little finger and ulnar half of the ring finger. When the median nerve is irritated, the numbness will be indicated on the palmar surfaces of the thumb and adjacent three fingers. In addition to these cardinal symptoms of pain and numbness, which appear early, various degrees of muscular weakness may occur. Paresis of the muscles in the ring and small fingers is often perceptible in cases of long standing. Actual muscle atrophy of the thenar or hypothenar eminences is a late sign.

The other symptoms are referable to *interference with the circulation of the arm*. The scalenus muscle or the extra rib may cause abnormal pressure upon the subclavian artery itself or upon its sympathetic nerve fibers.

which are situated in the lower cord of the brachial plexus. When such circulatory interference exists, the arm may be cool and pale or even purplish in color. There is diminution or obliteration of the radial pulse, which is especially well demonstrated by having the patient turn his head to the affected side and take a deep breath. A diminished blood pressure reading may be demonstrable on the affected side. Sometimes these vascular changes are detected only by oscillometric readings.

Although the extra rib may frequently be identified by palpation just above the middle of the clavicle, only a roentgenogram will demonstrate the extra rib with certainty.

The physician should be especially suspicious of a scalenus syndrome if the position and movement of the arm or neck modifies the symptoms. Elevation of the arm relieving them, depression of the shoulder accentuating them. Illustrative of this is the case reported from Ireland of a school teacher who was unable to grip a piece of chalk on a table, but was able to write easily on the blackboard. Conversely, during the World War it was recognized that some persons attempting to escape enrollment in the army were able to decrease or obliterate the pulse in one arm by certain movements of the shoulder.

Many patients learn to sleep with the arm above the head in order to obtain comfort. Extension of the head generally alleviates pain and other symptoms, forward flexion or turning the head to the affected side aggravates all of the symptoms. Even a deep inspiration may excite pain in the affected arm. Occupational positions which cause passive drooping of the shoulder tend to increase the compressive effect on the first rib and often aggravate pain, numbness, and coldness of the arm.

TREATMENT

The scalenus syndrome, whether mild or severe, is usually amenable to treatment. Even advanced cases with muscle atrophy may expect considerable improvement or actual cure. Fortunately, conservative measures help most cases. Operation is reserved for advanced or intractable cases.

The following are some of the conservative measures useful in the treatment of this condition. The use of an arm sling to relieve tension on the brachial plexus for several weeks may suffice. A figure of eight bandage about the shoulders, elevating the shoulder girdle, often gives relief. Shoulder raising postural exercises, which strengthen the trapezius muscle, may be used both for relief and for the prevention of recurrence. In some cases a traction apparatus, such as is described by Freiberg, may afford a cure without recourse to operation. With the patient in bed, traction is

applied directly to the elbows in the abducted position, light counter traction is applied in such manner as to be gripped by the hands

Prompt relief of pain is generally obtained from operation. Motor weakness of the hands, however, may not resolve for days or weeks after operation. The operative treatment consists of division of the anterior scalenus muscle after it has been retracted away from underlying structures. Due care must be exercised to protect the subclavian vessels and phrenic nerve which lie in intimate relation with the muscle.

Since there is danger of damage to the phrenic nerve and partial paralysis of the diaphragm in the course of operative manipulation, bilateral cases require a two stage operation. Only one side is operated at a time. If diaphragmatic function is normal after six or eight weeks, the second side may be operated on.

The presence of a true cervical rib, which produces symptoms and is demonstrable in roentgenograms, requires surgical treatment. If the scalenus anticus muscle is severed at its rib attachment, relief of pressure symptoms generally ensues, resection of the extra rib is usually unnecessary. There are cases, however, where surgical judgment at the time of operation dictates resection of the offending rib itself.

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CHAPTER XXXI

NERVE INVOLVEMENT AS A CAUSE OF PAIN IN THE CERVICAL SPINE, SHOULDER AND ARM

The close anatomic relationship between the large nerve structures and the bones in and about the spine makes it possible for disease or injury of the osseous structures to produce serious neurologic symptoms. Conditions primarily involving nerve tissues may be readily mistaken for arthritic disease and treated as such. In former years any painful condition about the arm, shoulder, or neck was all too commonly referred to as *neuritis* although the actual disease process may have involved a bone, a muscle, or a nerve. To a degree, the tendency to regard pain and *neuritis* as synonymous persists today, despite modern knowledge of pathologic processes and medical and surgical resources. The true understanding of the disease as well as the proper direction of therapy, demands a clear cut answer to the questions: Is this pain produced by a lesion of the spine, the nerve or the spinal cord? If so, what is causing it?

Although detailed examination of the nervous system is not called for in the routine examination of patients with arthritis, indications of neurologic disturbances obtained in the history should be followed through. Thus, any pain which is progressive or is present at one level calls for closer scrutiny of the nervous system. A history of numbness or other disturbances of sensation similarly demand confirmation by tests with light touch and pinpoint. Muscle weakness or muscle atrophy, when present should be viewed in reference to their central or peripheral nerve origin. Absent or exaggerated reflexes, especially of one side, point unerringly in the direction of nerve involvement. The information obtained by properly tapping the tendons with a reflex hammer will often resolve doubts about a case of neck or shoulder pain that has not yielded to treatment. Such study, including other corroborative tests, may disclose invaluable evidence of nerve involvement, previously unsuspected, in a patient presumably suffering from chronic arthritis.

As every practitioner is aware, the syndrome of pain and stiffness about the neck and shoulder with radiation down the arm, occurs frequently in people past forty years of age. Most of these cases are not instances of *'neuritis,'* as is so frequently diagnosed, but of *nerve root irritation*.

Radicular irritation frequently occurs in association with spinal arthritis

It may be caused by impingement of a bony spur or fibrotic tissue, perhaps edema or vascular changes. Pressure may be exerted directly on the sensory or motor nerve roots, or on the tiny blood vessels which supply



Fig. 109 Lateral view of cervical spine in hypertrophic arthritis showing marked degree of lipping at margins of vertebral bodies and narrowing of intervertebral spaces

them. The sensory root, being the larger of the two which pass through the foramen, is generally first to be affected by a constricting spur, or by irrita-

tion In the cervical region the posterior root is three times the size of the anterior Therefore the smaller anterior or motor root does not suffer as a rule until late Pain and sensory losses then are clinical signs of early



FIG. 110. Oblique view of cervical spine in hypertrophic arthritis showing narrowing at distal ends of intervertebral foramina.

involvement while well marked muscular weakness or actual atrophy is a late and infrequent sign In a series of 60 cases with cervical arthritis 39 had sensory symptoms only 4 motor (Bisgard)

The painful area in radicular irritation is segmental in distribution that is circular in distribution about the chest longitudinal in the arms and does not conform to the pattern of distribution of any peripheral nerve trunk. The painful segments are spinal segments corresponding with the affected intervertebral foramina. Two-thirds of all cases exhibiting root pains show roentgenographic evidence of hypertrophic arthritis with narrowing of the intervertebral foramina (Figs 109-110). In some roentgenographic proof is lacking because the pinching is caused by non opaque fibrous tissue. Neurologic confirmation however is sometimes present and should be sought for. Clinical proof of the extent and actual localization of such nerve involvement requires but a few moments directed to the simple facts of nerve distribution and is desirable both for diagnosis and for effective treatment.

Sensation is carried in the back of the head from vertex to the neck by the second spinal (cervical) nerve over most of the neck by the third cervical in the shoulder by the fourth. If the arm is held out thumb upward—as in pistol shooting—the other sensory segments may be simply followed in rotation by going around the arm thus:

From shoulder to thumb	Fifth cervical
From thumb to ring finger	Sixth and seventh cervical
The ulnar side of ring finger the small finger and around the elbow	Eighth cervical and first thoracic
From elbow under arm axilla and precordium	Second and third thoracic

This rapid and practical method of orientation serves well in spotting the exact site of the trouble—that is whether it is in the higher or lower portions of the neck. Pain which is referred to the back of the head the suboccipital region the neck or along the upper or thumb-side of an arm outwardly extended comes from the upper part of the neck (C 2-C 5). Pain referred down to the little finger along the ulnar side is lower in origin (C 8-Th 1). The pain of arthritic disease for example is very frequently localized in the upper cervical segments and referred along the neck back of the head and thumb-side of the outstretched arm (C 2-C 5) that of angina pectoris shoots along from precordium to the little finger (C 8-Th 1). In elderly patients attention to these nerve distribution zones may give the initial clue to the real nature of the complaint and the effort of further neurologic confirmation is then well repaid.

Simple tests for tactile sensation for pain and for heat and cold furnish the necessary evidence of nerve impairment. The loss of a reflex—

biceps or triceps—is a stronger bit of evidence. Signs of muscular weakness or of atrophy of the muscles making up the ball of the thumb or the corresponding hypothenar eminence, with coldness and blueness of a member, offer conclusive proof. With this evidence gathered, one may proceed towards solving the problem in hand, that is, whether the given nerve symptoms are due to arthritic narrowing of intervertebral foramina, which produces pinching of the spinal nerve roots or blood vessels (spelling radiculitis), or by another, perhaps far more serious involvement of nerve tissue.

To be sure, not every case of spinal arthritis is painful. In fact, evidence of the primary disease may be minimal or absent, while the symptoms due to nerve root irritation may be both annoying and varied.

In typical form, complaints due to nerve root irritation in the cervical region are familiar enough. Over half of those who suffer from arthritis of the cervical spine complain of "headache" referred to the occiput. This type of algosia, familiar to practitioners as the "indurative" or "rheumatic" type of headache, has already been described (page 274). A common complaint, too, is a feeling of heaviness in the shoulders, with or without soreness and stiffness in the neck and between the shoulder blades.

Tingling, numbness, or anesthesia of the fingers, often accompanied by pain shooting down the arms, are equally typical and even more confusing, yet the occurrence of these root phenomena is so frequent and so practical a problem as to tax the diagnostic and therapeutic resources of the physician in almost every day's work. The necessity of differentiating arthritic manifestations from primary nervous disease becomes more urgent with the increasing severity of symptoms. It is well, therefore, to bear in mind both the original and counterfeits of serious arthritic nerve involvement.

As we have just indicated, the intervertebral foramina, from which the spinal nerves emerge, are vulnerable, especially in persons past middle age. Chronic arthritis of the spine may give rise to radiculitis of such severity as to resemble the symptoms of other, well known primary nerve disorders. Hypoesthesia, anesthesia, weakness, or paresis of the arm, various grades of incoordination, and absence of reflexes have been known to occur secondary to nerve root damage by spinal arthritis, without primary disease of the cerebrospinal axis itself. Occasionally, atrophy in the small muscles of the hand or arm is so advanced as to present the suspicion of syringomyelia or progressive muscular atrophy. Moreover, the acts of coughing or sneezing, as well as compression of the jugular veins, may precipitate or aggravate the pain even in radiculitis, hence caution is required in evaluating the picture presented, lest one interpret these as signs of intraspinal disease.

The therapeutic measures applicable to the treatment of cervical hypertrophic arthritis have already been discussed (page 283).

NON-ARTHRITIC CONDITIONS WHICH MAY CAUSE PAIN IN THE SHOULDER GIRDLE AND ARM

Without going into the minutiae of neurologic diagnosis we may here advantageously consider the nerve lesions which may, in some circumstances be confused with spinal arthritis

Peripheral Neuritis

Peripheral polyneuritis, due to intoxicants such as alcohol and lead, or to vitamin B deficiency, is often bilateral or actually symmetrical in the two extremities. The affected nerve itself is tender to palpation along its course, it is sometimes felt as a thickened cord in its more superficial portions. All forms of sensation—touch, pain, temperature and sense of position—are equally diminished, or lost. The deep tendon reflexes become less active or disappear. Muscle atrophy and vasomotor disturbances may develop. Generally motor weakness exceeds sensory loss in peripheral neuritis, if not it and sensory loss are equally affected. A history of dietary deficiency or of exposure to cold, trauma, or toxic agents, aids in diagnosis. Most important, the symptoms and signs are limited to the regions of distribution of a given peripheral nerve, as the radial, median, or ulnar.

In contrast, radiculitis produces a relatively extensive area of pain, generally radiating from the spine out to the distal portion of the hand. If one or two intervertebral foramina on one side are involved the resulting radiculitis is of course unilateral, and produces a longitudinal area of diminished or lost sensation extending beyond the boundaries of distribution of any known peripheral nerve. Moreover, in radiculitis there is no loss of motor function unless the anterior (motor) roots are also involved. Pure sensory radiculitis is the rule, whereas pure sensory polyneuritis is rare.

Neurofibroma

The intervertebral foramina are also vulnerable sites for the location of neurofibromata—benign tumors arising from connective tissue of the nerve. These are frequently multiple, and extremely painful, often producing lancinating neuralgias. Neurofibroma is more often a cause of severe sciatica than of brachial neuralgia, since the nerves of the cauda equina are favorite sites for their occurrence. Still, they should be considered when severe pain in the arm is a symptom. They are benign and yield to neurosurgical treatment.

Pressure Upon Brachial Plexus

Pressure upon the brachial plexus gives rise to similar symptoms. Such pressure may, for example, be exerted in the neck region by a cervical rib.

or abnormal scalenus muscle or even by a sulcus tumor at the apex of the lung

Disease in the Spinal Vertebrae

The vertebrae themselves may be potential sources of trouble. *Intervertebral disk herniations* old or unsuspected *spinal fractures* and *metastatic carcinomatous infiltration* into the spine need only mention at this point. The latter condition is especially apt to produce severe intractable pain requiring morphine for relief. It is well to be alert to the possibility of new growth in every case of rheumatism requiring morphine.

Compression of Spinal Cord

Compression of the spinal cord itself is a serious consideration. If one is guided by sensory losses alone, signs of such compression may be missed inasmuch as the involvement of only one sensory segment may produce no disturbance that can be detected; the adjacent sensory segments overlap and maintain normal sensation in a single involved root area. *Hypoesthesia* (diminished sensibility) is found more often than frank anesthesia. Percussion over the spine may reveal a tender spot while lateral pressure on the spinous processes may disclose a painful area over the region where the cord is compressed. Often a zone of hyperesthesia or touchiness will be evident at the level of the lesion. The effect of coughing or sneezing in aggravating the pain of cord compression is already well known.

Spinal fluid study including protein determinations and manometric examination for block as well as specialized neurologic study of sensory levels, sweating level and even *lipiodol injection* may be required before an accurate diagnosis is possible in cases in which there is serious suspicion of cord compression.

Chronic Hypertrophic Pachymeningitis

Affections of the meningeal coverings of the cord are infrequent but occasionally encountered causes of pain. *Chronic hypertrophic pachymeningitis* an inflammation of the dural covering is most frequent in the cervical region. Marked involvement of the sensory roots of the nerves that make up the brachial plexus takes place and pain in the shoulder girdle or arm ensues. Such pachymeningitis may be caused by syphilis, tuberculosis, trauma and perhaps by alcoholism. The symptoms are generally bilateral. Coughing or straining produces pain or aggravates it while percussion elicits tenderness over the involved area of the spinal column.

Other Affections

Adhesive arachnoiditis (circumscribed serous meningitis) may produce similar signs of pressure on the cord. *Hemorrhage* into the spinal cord

thrombosis of the spinal vessels, or arteriosclerotic "claudication" of the cord, while rare must occasionally be taken into consideration

The spinal cord itself may be the site of a disease whose voice is 'pain and stiffness'. Neurosyphilis, of course, heads the list of such conditions. Syphilis of the nervous system has a great tendency to involve the sensory roots of the spinal nerves as well as the posterior columns of the cord itself. It is more likely to affect the lower part of the cord than the cervical region. The characteristic changes in the pupillary reflexes, as well as the serologic tests, may provide the clues to the real disorder. Radiculomyelitis of infectious or toxic origin is now recognized clinically, and its presence may be confirmed by spinal fluid studies. Encephalitis and encephalomyelitis, especially during epidemics of these diseases, have been ushered in slowly by pain in the back or stiffness of the neck. These are the so-called 'neuritic' forms of these serious conditions, and are due to meningeal and root irritation that accompanies invasion of the nervous system. The diagnosis in such cases is resolved within a short time by the spinal puncture and by the clinical course of the disease.

There are other miscellaneous conditions that deserve mention as a cause of pain in the neck or arm. Occupational cramp is frequently encountered. If it is not corrected after several days' rest, it should arouse suspicion of organic disease. Herpes zoster may cause puzzling pain for several days before the eruption appears. Referred pain in the shoulder from irritation of the diaphragm by pulmonary or abdominal disease should be recognized during the course of general examination. Psychalgias in the neurotic or constitutionally inferior must be considered only after the exclusion of organic causes.

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PART VIII

LOW BACK AND SCIATIC PAIN

LOW BACK AND SCIATIC PAIN SOME GENERAL CONSIDERATIONS

CERTAIN ANATOMIC AND PHYSIOLOGIC RELATIONSHIPS BEARING ON THE
PATHOGENESIS OF LOW BACK AND SCIATIC PAIN

SOME METHODS OF TREATMENT FOR LOW BACK AND SCIATIC PAIN

CAUSES OF LOW BACK AND SCIATIC PAIN CLASSIFICATION

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN MUSCULAR AND
LIGAMENTOUS STRAINS AND SPRAINS

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN POSTURAL STRAIN

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN ARTHRITIS
FIBROSITIS (MYOFASCITIS)

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN CONGENITAL
ANOMALIES

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN ABNORMALITIES
AT THE SACRO-ILIAC JOINTS

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN AFFECTIONS OF
THE PYRIFORMIS MUSCLE

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN CONTRACTURE
OF THE ILLIOTIBIAL BAND

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN ABNORMALITIES
AT THE VERTEBRAL ARTICULAR FACETS

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN PROTRUSION OF
LUMBAR INTERVERTEBRAL DISCS

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN THICKENED LIGA-
MENTA FLAVA

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN TUMORS— RHEU-
MATISM REQUIRING MORPHINE

CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN MISCELLANEOUS
Fractures

Dorsolumbar Sprains

Pressure from Lesions in the Pelvis and Rectum

Constitutional Diseases

Tonic Factors

Primary (Idiopathic) Sciatic Neuritis and Herpes Zoster

Vascular Diseases

Functional Nervous States

Malingering

CHAPTER XXXII

LOW BACK AND SCIATIC PAIN

SOME GENERAL CONSIDERATIONS

Many patients with pain in the lower back and along the course of distribution of the sciatic nerve come to the physician literally clamoring for relief. But in their anxiety to obtain help quickly, they choose too often the longest, most circuitous route, that from physician to chiropractor, from osteopath to naturopath, and then back again to the physician.

Relief of these conditions is beset with obstacles. so many different etiologic factors may be involved in different cases. Opinions as to causes and treatment are widely divergent. These inherent difficulties are accentuated by the too frequent failure to be guided by an etiologic diagnosis in the discriminating selection of therapeutic measures. Low back and sciatic pain, like tachycardia, is obviously only a symptom of some underlying pathologic process. Unless the basic cause of the condition is discovered, treatment is merely a 'shot in the dark' and just as likely to miss its aim. Although this fact has been stressed repeatedly by all writers on this subject it has not yet served invariably as a guiding principle to treatment.

No doubt the difficulty of establishing an etiologic diagnosis in these cases has thwarted enthusiasm for attempts to do so. Certain gaps in our knowledge concerning pathogenesis have been disconcerting. There is, moreover, no sure and easy way to treat some of these conditions, even when the therapeutic approach is seemingly cleared by an etiologic diagnosis. Despite all these handicaps we have made tremendous strides, in recent years, in crystallizing knowledge concerning certain specific causes in many cases of low back and sciatic pain, and in reconciling apparently conflicting views on the subject. We have been able to relieve completely, sometimes spectacularly, so many cases formerly dismissed as discouraging and intractable, that we have come to realize that every attempt at careful investigation of every case is mandatory.

The number of patients afflicted with low backache or sciatic pain, or both, is astonishingly large. Almost invariably they seek first the advice of their family doctor. It is well that it is so, for a large proportion of them can be dealt with satisfactorily by the general practitioner if treatment

of a normal physiologic status than upon restoration of normal anatomic structure. The latter is in fact, rarely possible to achieve when pathologic changes have already become established.

Various etiologic factors causing low back or sciatic pain will be discussed separately in subsequent chapters. This does not invalidate the views just expressed that in a given case a multiplicity of interrelated etiologic mechanisms, and not merely a single specific lesion, is usually involved.

GENERAL CHARACTER OF THE SYMPTOMS AND THEIR DIAGNOSTIC SIGNIFICANCE

Regardless of the underlying pathologic lesion, the symptoms associated with abnormalities at the lower back are similar in their general pattern. They may be grouped arbitrarily as manifestations confined to the lower back or along the course of distribution of the various roots of the lumbosacral nerves or as combinations of the two.

Pain in the lower back may vary widely in intensity and general character. It may manifest itself as only a dull ache, as a sensation of heaviness, as stiffness or it may be a sharp severe pain, at times agonizing requiring opiates for relief. It is remarkable that the severity of the symptoms is not necessarily proportionate to the extent of the causative pathologic change. An acute strain of a back subject to chronic postural muscular overtension may produce violent pain although the roentgenogram may be entirely normal. A lumbar spine grossly damaged by osteoarthritic changes may on the other hand produce only a dull ache or no symptoms at all.

The situation of the pain is not a very reliable index of the site of the causative lesion. I have seen pain predominantly in the sacro-iliac region proved to be the result of lumbosacral joint disease. Pain confined to the lumbosacral area is however more likely to be the result of a lesion there. Tenderness is more reliable as a localizing sign.

Frequently the pain is referred to regions beyond the back, the posterior aspect of the hip, buttock and thigh or the posterolateral aspect of the thigh, calf and foot. Less frequently the backache is associated with pain in the anterior thigh or groin.

The situation of the referred pain and its distribution may be of greater aid in localizing the probable site of the primary lesion than the backache. This fact applies particularly to pains caused by pressure on specific spinal nerve roots or by inflammatory changes in them. Such pains, tracing the segmental distribution of the affected roots are likely to be sharp, burning or stabbing in character. Although not infrequently constant in severity,

regardless of rest or activity, they are in some cases temporarily relieved by walking

Low back pain with classical "sciatica" is a syndrome only too well known. A racking pain anywhere in the lower back, aching and tiredness, and frequently soreness and stiffness may be noted. In addition there is a burning or gnawing pain of variable severity, spreading downward along the back of the thigh and calf or along their posterolateral aspect. It may not extend beyond the knee, or it may reach all the way to the ankle frequently to the outer malleolus, and even to the toes. A sharp, knifelike pain which shoots down the back of the thigh or the whole leg, may be so severe as to rouse the patient from sleep or to prevent him from sleeping.

In addition to pain in the back and along the thigh and leg, there may be a "drawing" or "pulling" sensation in the leg or ankle, or a feeling of "constriction." These are generally caused by nerve root irritation rather than by muscle spasm. Such discomfort is more often associated with low lumbar or lumbosacral than with sacro iliac disease.

A dull, aching discomfort in the lower back, associated with stiffness and soreness, particularly early in the morning, and waning as the day goes on, suggests arthritis or fibrositis as the causative factor.

The mode of onset of the pain may yield some clue as to the possible etiology. The importance of a history of trauma is obvious. The severity of the trauma and the manner in which it was exerted (either directly on the suspected joint or indirectly, as by twisting) are all important factors. However, traumatic strain of the back may initiate symptoms of pre-existing chronic arthritis, previously symptomless.

The previous history with reference to the occurrence of acute infections, nonspecific or specific in character, and of rheumatic manifestations in joints of the extremities, may cast some light on the possible nature of the low back condition.

The history with reference to the urinary tract (the prostate in men, and the gynecologic history in women) may yield important clues for further investigation. Such conditions in the pelvis constitute, however, insignificant factors in low back pain.

The history with reference to the occurrence of fatigue and nervous exhaustion preceding the onset of the backache may yield additional clues as to possible predisposing or aggravating influences. These may have an important bearing on subsequent treatment.

It is well to ascertain the patient's reaction to his condition. He may be one of those constitutionally inadequate personalities to whom "ill health" is temporarily vital, or he may be involved in litigation or be receiving insurance or "compensation" benefits, factors which, in certain types of individuals, may block every effort of patient and physician in attaining

recovery. It is necessary to evaluate properly such influences on the disability presented. It is particularly well to remember that a patient such as we have just described is not a malingerer, and with proper handling may get well.

THE GENERAL MEDICAL INVESTIGATION

The general medical investigation is as important in the study of patients with low back pain as in those with any other condition. An examination confined to the back may not reveal the most glaring facts necessary for diagnosis. On several occasions the finding of a hard lump in the breast in the course of the general examination indicated the correct presumption of metastatic carcinoma as the cause of severe backache, when examination of the spine was inconclusive. Myotic, static pupils may indicate the cause of bilateral sciatic pain, and so on. Observation of changes in the reflexes of the legs, the presence of hypoesthesia or hyperesthesia, may clearly reveal segmental nerve root involvement. It may suggest the necessity for detailed neurologic study and examination of the spinal fluid, and perhaps myelography, which may alter radically the final diagnosis as compared with the initial impression.

THE EXAMINATION OF THE PATIENT WITH LOW BACK AND SCIATIC PAIN

Methodical and complete examination of the back and extremities aids in determining the site of the lesion and, possibly, its etiology. Smith Petersen (1924) described many of the objective signs which aid in distinguishing between lumbosacral and sacro-iliac lesions. Although helpful in most cases, these signs are not always so clear-cut as to be entirely reliable in establishing the site of the lesion in every case.

Inspection. The postural attitude of the body should be noted, including deviations from normal in the lumbar curve and the alignment of the feet. In acute cases flattening of the lumbar curve and even kyphosis, with spasm of all the muscles about the lower back, may be present. In some instances the muscle spasm produces a list of the spine. The scoliosis may be directed either away from or toward the side affected. The list is of little help in diagnosis for, even with predominantly unilateral involvement, there is no consistent relationship between the direction of the list and the side affected. On the whole, then, listing is of little localizing value. It does, however, indicate the existence of muscle spasm and imbalance and is therefore an indication of the acuteness of the process.

Inspection of the spine may reveal marked lordosis and associated find

ings (described in detail later) suggestive of spondylolisthesis. Inspection includes, of course, observation of the body type and attitude and of the muscular tone and development. Mere inspection may also reveal complete flattening or kyphosis caused by various types of destructive lesions of the anterior portions of the vertebral bodies.

Palpation. Palpation may confirm a previous suspicion of muscle spasm or may indicate its existence when it is not sufficiently marked to be noted on inspection. Palpation of the spine also elicits certain details of its configuration, particularly with regard to the alignment of the vertebrae. Local tenderness elicited by palpation is of considerable importance in determining the exact site of the pathologic process. Superficial tenderness generally indicates muscular rather than ligamentous or joint involvement.

In lumbosacral lesions, tenderness is most marked or present only directly over the lumbosacral junction, between the spinous processes of the fifth lumbar and first sacral vertebrae, or just lateral to that corresponding to the location of the lumbosacral articular facets, or just mesial to the posterior crest of the ilium, corresponding to the point of origin of the ilio-lumbar ligaments. In sacro iliac conditions, tenderness is more apt to be localized to the inferior sacro iliac ligaments and along the upper border of the sacrosciatic notch.

Tenderness over the sciatic nerve is thought to be indicative of involvement of either its trunk or roots, but may also be caused by muscular involvement. Such tenderness is generally elicited best at the level of the gluteal fold, but may be perceptible also at the posterior aspect of the thigh. It is not of value in differentiating between lumbosacral and sacro iliac lesions but, when present unilaterally, does serve to indicate the side involved.

Motions. Analyzing the effect of motions at the spine, with the patient standing, sitting, and lying, adds data of additional value in confirming the site of involvement.

Motions standing. Flexion of the lower spine may fail to produce the posterior rounding of the lumbosacral curve, as in cases of spondylolisthesis. The extent of flexion may of course be greatly limited, either by pain, muscle spasm, or ankylosis. It is important to determine, through collateral evidence, which of these factors is responsible.

In lumbosacral conditions, the lumbosacral segments may be held perfectly rigid by muscle spasm. In that case, forward bending takes place either in the upper lumbar or dorsolumbar regions, or at the hips. Extension at the lumbosacral junction is equally limited, the patient therefore attempts to bend backward by flexing his knees. Lateral flexion at the lumbosacral junction is equally impossible; consequently, side bending takes place at the dorsal or upper lumbar levels. It is logical to expect, of

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course, as is actually the case, that lateral flexion is generally freer in the direction away from the side affected than toward it

In sacro-iliac conditions the patient may bend forward by flexion of the lumbar spine then by tilting the pelvis until the hamstrings become taut Motion is impeded at this point because of pain To bend further, the patient may attempt to relax the hamstrings by bending the knee on the affected side Backward bending is quite free at the lumbosacral junction Lateral flexion in either direction may be limited, but more frequently the range of motion is wider toward the affected side than toward the other, although not infrequently, the reverse is true

Motions sitting In the sitting position the hamstrings are relaxed and leverage is not transmitted to the pelvis Consequently, the patient with sacro-iliac disease (in whom forward flexion in the standing position is markedly limited) may bend forward with a remarkable degree of facility while sitting The patient with lumbosacral disease is equally unable to bend forward sitting or standing

Motions lying Passive flexion of the lumbar spine, by flexing the hips and knees is markedly limited and painful when the lesion is in the lumbosacral region Since there is no leverage transmitted to the pelvis by such passive flexion of the spine, such motion is apt to be more free in sacro-iliac cases Obviously in acute sacro-iliac disease such motion may induce some discomfort which though limiting motion, does so to a much lesser extent than active flexion with the patient standing

Of course mixed lesions are so common that various combinations of response to the described movements are frequently seen

SPECIAL TESTS

Straight leg raising (Lasegue sign) The explanation of the mechanism producing limitation of straight leg raising is disputed Although limitation of straight leg raising almost invariably occurs when there is involvement of any of the roots of the sciatic nerve, it may take place also, as will be indicated later with acute muscular pains, in the absence of direct involvement of nerve roots Treiberg and Vinke, and others are of the opinion that the Lasegue sign may be produced by spasm of the psoas muscle induced by a variety of circumstances

A positive Lasegue sign is of diagnostic value by indicating first the possibility of nerve root irritation, although it does not establish that fact unequivocally When the Lasegue sign is positive on one side and negative on the opposite it may be inferred that one is dealing with a unilateral lesion Some writers have interpreted such a finding as indicative of sacro-iliac, in contradistinction to lumbosacral, disease This is not the case,

however Although lumbosacral lesions are apt to induce bilaterally equal positive, *Lasegue* responses, there are so many exceptions that the significance of this test in differentiating between lumbosacral and sacro iliac involvement is invalidated

Smith Petersen interpreted the straight leg raising test on the basis of leverage transmitted through the hamstrings to the pelvis He advised that in doing straight leg raising slowly, one hand should be under the patient's lower spine As the hamstrings tighten, leverage is gradually applied to the side of the pelvis If pain is brought on before the lumbar spine begins to move I feel that this reaction is definitely in favor of a sacro iliac condition If, however, pain does not come on until after the lumbar spine begins to move, this reaction is in favor of either a sacro iliac or a lumbosacral condition, because the sacro iliac joint is under leverage and the lumbar spine is moving ' For reasons stated such interpretation of the *Lasegue* phenomenon is not universally applicable

Gaenslen test Hyperextension of one thigh while the sacrum is fixed produces pain on that side when there is involvement of the corresponding sacro iliac joint The means of eliciting this sign will be described in greater detail when lesions of the sacro iliac joint are discussed (page 473)

The *Ober* sign Maintained abduction of the leg presumed to be pathognomonic of contracted fascia lata will be described later under the latter topic

Compression of the crests of the ilia may induce pain if there is an active, acute condition affecting a sacro iliac joint Since the lumbar spine is not affected by this maneuver, pain does not result in lumbosacral lesions Nor will such compression necessarily induce pain in a sacro iliac condition if the process is not acute The value of this procedure in differential diagnosis is, therefore, limited To carry out this test the patient lies on his unaffected side The examiner puts his forearm on the affected side, leaning on it with sustained pressure for as long as half a minute The pain which results when the test is positive is of course, the result of leverage transmitted through the sacro iliac joints

Rectal examination in addition to revealing other types of relevant data may also elicit tenderness over the lower end of an involved sacro iliac joint but only when the process is inflammatory

ROUTINE LABORATORY INVESTIGATION

The routine laboratory investigation of such patients may yield data of crucial importance for diagnosis The necessity for routine serologic investigation for syphilis has only recently been emphasized as it should be To say the least, a blood count may hasten diagnosis It may, for example,

reveal evidence of myelogenous leucemia which, by leucocytic infiltration around the lower lumbar nerve roots, may produce sciatic pain. Such a diagnosis would not only indicate the true prognosis, but would suggest appropriate roentgenotherapy which might afford much desired, even though temporary relief of symptoms. The finding of Bence Jones protenuria would be of equal importance in the diagnosis of myeloma.

ROENTGENOGRAPHIC INVESTIGATION

Roentgenographic investigation is of importance in the accurate diagnosis of all cases of low back disability, in some it is all important. A normal roentgenogram has definite value. It may confirm the impression that the pathologic condition is either outside the bony structures of the spine or that it is still in its early stages. In a case presenting segmental nerve root pains along the leg with or without low back pain a normal roentgenogram of the spine dictates the necessity of making sure that no intraspinal source of nerve root pressure or irritation exists.

Abnormal findings in the roentgenogram may localize the site and indicate the nature of the underlying pathologic process. Such findings may reveal a totally unexpected pathologic basis for the symptoms. The characteristic osseous changes of Paget's disease, the osteoporosis of hyperparathyroidism or bone destruction of metastatic carcinoma, may cast an entirely new light on the clinical picture. That such conditions are among the more rare causes of low back pain does not justify postponement of their diagnosis until all of the obvious signs appear.

Roentgenograms of the spine, to be of value in diagnosis, must conform to certain minimal standards. The quality of the roentgenogram must be sufficiently good to permit objective interpretation. Roentgenographic diagnosis of low back conditions is attempted too often from roentgenograms that are totally inadequate in one respect or another. An anteroposterior view of the spine will not reveal many pathologic conditions which might easily be seen in a lateral view. Anteroposterior and lateral views represent minimal requirements of adequate roentgenologic study of the low back, often oblique views are desirable in addition. And, of course, correct interpretation of the roentgenologic observations is important. We must also seek to establish a logical relationship between the roentgenographic abnormalities noted and the clinical findings.

NEUROLOGIC STUDY, SPINAL FLUID EXAMINATION, AND MYELOGRAPHY

It has been recognized for a long time that tumors of the spinal cord may produce symptoms resembling the more usual types of sciatic pain.

The necessity for spinal fluid examination and myelography in certain of these cases has, therefore, come to be appreciated. In the past few years special emphasis has been accorded the neurologic aspect of these conditions. It has become evident that such study—at least cursory neurological examination—should be carried out in every such patient. On such neurologic study depends the detection not only of intraspinal neoplasms, but also of intervertebral disk protrusions, thickening of ligamenta flava in inflammatory affections of the meninges, lesions of the facets—a group of conditions recognized to be of considerable importance in relation to sciatic pain. The neurologic examination gives the first hint of the possible existence of these lesions; this must then be established by examination of the spinal fluid, and in some cases, by myelography with iodized oil. The more specific indications for such studies will be discussed in subsequent chapters.

THE THERAPEUTIC TEST

As in other phases of clinical medicine, the therapeutic test may have to be invoked for confirmation of the diagnosis in some of these cases. The association of poor posture and localized lumbosacral arthritis, for example, may cause some difficulty in determining which of these two factors is largely responsible for the symptoms. Eliminating the postural error, when possible, and observing the effect of this therapeutic procedure will probably clarify the issue.

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CHAPTER XXXIII

CERTAIN ANATOMIC AND PHYSIOLOGIC RELATIONSHIPS BEARING ON THE PATHOGENESIS OF LOW BACK AND SCIATIC PAIN

The precarious anatomic situation of the lumbosacral junction renders it peculiarly vulnerable to a variety of pathologic processes. Constituting the point of maximum lordosis, the commonest site of congenital aberrations, and at the same time carrying the heaviest load, the lumbosacral articulation is extremely susceptible to every type of injury, including physiologic and accidental trauma, secondary infectious processes, and so forth.

RELATIONS BETWEEN THE LOWER BACK AND THE ROOTS AND TRUNK OF THE SCIATIC NERVE

It is impossible to understand the pathologic and clinical implications of disease in the lower back without a vivid mental picture of the anatomic relationships between the lumbar spine, the lumbosacral and sacro-iliac articulations, and the nerve roots and trunk of the sciatic nerve. These relationships were brought out most strikingly by the anatomic observations of Danforth and Wilson, from whose report we shall quote extensively.

The sciatic nerve, we recall, is derived from the fourth and fifth lumbar and the first, second, and third sacral roots. The *peroneal* or *external* popliteal division of the sciatic nerve comes from the fourth and fifth lumbar and the first and second sacral roots. The *tibial* or *internal* popliteal division of the sciatic nerve is derived from the fourth and fifth lumbar and the first, second, and third sacral roots. The superior gluteal nerve comes from the fourth and fifth lumbar and the first sacral roots. The obturator and the femoral or anterior crural nerve come from the second, third, and fourth lumbar roots. The segmental distribution of the peripheral cutaneous nerves in the lower extremity is shown in the accompanying diagrams (Fig. 111).

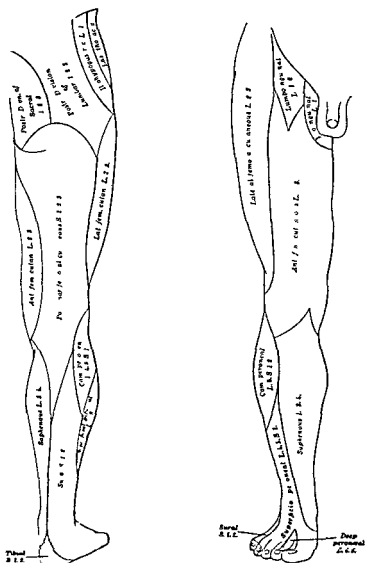


FIG. 111. Diagrams indicating the segmental distribution of the cutaneous nerves of the lower extremity. Anterior and posterior views. (From Gray's Anatomy, Lett & Ichiger.)

Although the gross anatomic features of the lumbar spine and of the sacro iliac joints are in general well known, it is important to emphasize certain anatomic relationships between the nerve roots of the lumbosacral

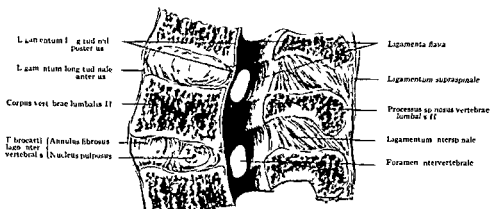


FIG. 112. Sagittal section through the spinal column in the lumbar region to show the vertebral canal and the intervertebral foramina (from Callander's Surgical Anatomy W. B. Saunders Co.)

plexus and the related skeletal structures that have a bearing on the production of sciatic pain.

RELATIONS BETWEEN LUMBAR NERVE ROOTS AND THEIR INTERVERTEBRAL FORAMINA AND CANALS

On emerging from the neural canal, the nerve roots proceed through the intervertebral canals, which, as will be revealed presently, are one of the sources of sciatic root irritation. The roof of the intervertebral canal is formed by the inferior intervertebral notch of the vertebra above, its floor by the superior intervertebral notch of the vertebra below. Its anterior margin is formed by a part of the vertebral body above, the intervertebral disk, and the superior portion of the vertebral body below. Behind lies the posterior articulation (Fig. 112).

Danforth and Wilson emphasized the relationship between the sizes of the lumbar and lumbosacral intervertebral foramina and canals and the sizes of the nerve roots passing through them. They found 'the foramen between the fifth lumbar vertebra and the sacrum always the smallest, that between the fourth and fifth vertebrae the next larger, and that between the third and fourth usually the next, although sometimes the second and third were about equal. Quite contrary to the size of the foramen or canal, is the size of the nerve root enclosed. The fifth was always the largest, the fourth next to the largest, and the third next smaller as a rule, although sometimes the second and third roots were about equal in size. Otherwise

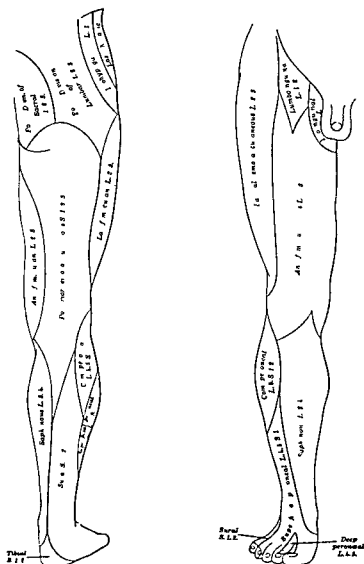


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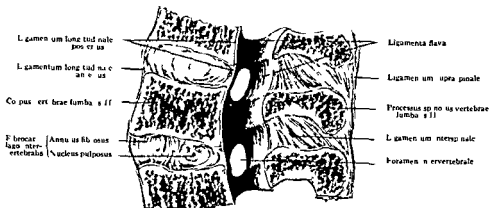


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expressed, the largest nerve root (the fifth) always had the smallest canal, and frequently it almost filled the canal, while the fourth rarely filled the opening and the second and third never" (Fig 113)

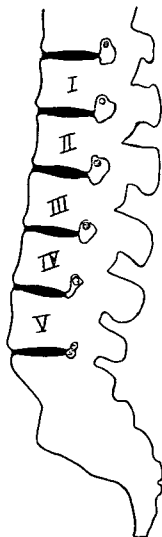


FIG 113 Drawing illustrating the relative sizes of the intervertebral canals of the lumbar spine, also the sizes of the corresponding nerve roots (After Danforth and Wilson, *Journal of Bone and Joint Surgery*, 7 114 1925)

It should be recalled, also, that throughout its course in the intervertebral canal the nerve root is surrounded by an arterial and venous plexus and by a small amount of fibro-areolar tissue. Nerve root irritation is possible, then, under the many conditions which may lead to encroachment upon the barely adequate lumen of the fourth and fifth lumbar intervertebral canals.

Putti described in detail various anomalies of the articular facets in the lumbar and lumbosacral regions, which may have the effect of altering the shape and reducing the capacity of the intervertebral foramina. By altering the mechanics of the spinal column, they may induce a localized arthritis, which itself may irritate the nerve trunk. Any inflammatory condition in the vertebral articulations (between the articular processes), leading to swelling or congestion in the venous plexus surrounding the nerve root would also encroach upon the already crowded condition within the canal producing pressure on the nerve involved and thus, pain referred along the course of its distribution. We shall revert to this phase of the subject again when we discuss in greater detail the relation of disease in the articular facets to low back and sciatic pain.

RELATIONS BETWEEN LOWER LUMBAR NERVE ROOTS INTERVERTEBRAL DISKS, AND SACRO ILIAC JOINTS

Danforth and Wilson also stressed the intimate relationship of the fourth and fifth lumbar nerve roots to the intervertebral disks at the last interlumbar and lumbosacral junctions respectively.

'The fourth lumbar nerve after leaving the intervertebral foramen lies closely in contact with the lateral surface of the intervertebral disk between the fourth and fifth bodies and their articular margins, and descending lies on the anterior surface of the transverse process of the fifth. From its position on the transverse process, it continues downward across the space between the process and the sacrum, to lie on the ala and anterior surface of the sacrum. In its course on the sacrum it always lay mesial to the sacro-iliac joint until it reached the great sciatic notch, where it was sometimes mesial to it and sometimes lay directly on the joint' (Fig 114.)

It is apparent that nerve pain due to involvement of the fourth root, either from position or from pathologic change in the joints, might occur (1) in its course in the intervertebral canal, (2) at the lateral margin of the joint between the fourth and fifth lumbar bodies, or (3) at the lower margin of the sacro iliac joint, at the great sciatic notch.

Danforth and Wilson also stressed the intimate relationship of the fifth lumbar nerve root to the intervertebral disk. 'The fifth root after emerging from the foramen between the fifth lumbar body and the sacrum lies in direct contact with the lateral margin of inferior surface of the fifth, with the intervertebral disk, and with the lateral margin of the superior articular surface of the sacrum.' Continuing downward, they added, the fifth nerve lay, always in our dissections, well mesial to the sacro-iliac joint, until it reached the great sciatic notch. Here it was usually mesial to the joint, but very close to it, and occasionally lay on the joint."



FIG. 114. Drawing from dissection showing the relations of the sacral plexus. Note especially the course of the fourth and fifth lumbar nerve roots and their relations to the fifth lumbar intervertebral disk and to the sacroiliac joint. (After Danforth and Wilson *Journal of Bone and Joint Surgery* - 11, 19-5)

It is therefore easy to understand why sciatic pain over the area of distribution of the fifth lumbar root might occur either (1) in its course through the *intervertebral canal*, (2) *at the lateral margin of the joint between the fifth lumbar vertebra and the sacrum* and (3) *at the lower margin of the sacro iliac joint* (Fig 114). It should be remembered, however, as Danforth and Wilson have emphasized, that the ligaments holding the fifth lumbar vertebra to the ilia and sacrum are very strong, confining the fifth lumbar nerve to a relatively narrow space in closest proximity to the lumbosacral articulation. In contrast, there is nothing at the lower margin of the sacro-iliac region to hold the nerves against the joint. Hence the case with which the nerve may be displaced by pathologic lesions occurring there. For these reasons Danforth and Wilson concluded that "the chances for nerve involvement are much greater in the lumbosacral region than in the sacro iliac region."

This view, which has received acceptance from most critical students of the subject, must be considered by any one seeking a rational explanation for sciatic pain associated with pathologic processes in the lower back.

PERIPHERAL INNERVATION OF THE STRUCTURES ABOUT THE LOWER SPINE IN THE PATHOGENESIS OF LOW BACK AND SCIATIC PAIN

As the previous discussion has indicated, the nerve roots of the lumbosacral plexus and the articulations of the lower lumbar spine are in such close anatomic relationship that sciatic pain may easily result from direct pressure on nerve roots by pathologic lesions in proximity to them. Many cases of low back and sciatic pain, however, are not the result of direct pressure on nerve roots. Among the most common causes of pain in the distribution of the sciatic nerve are affections of muscular and ligamentous structures about the lower back and pelvis.

Localized pain in those areas is readily explained. Branches of the posterior divisions of the lumbar spinal nerves may be irritated by pathologic processes and convey sensory impressions of pain from the muscles of the back, the ligaments, aponeuroses, and the periosteal attachments at the intervertebral and sacro iliac articulations, as well as from those joints themselves. As a result of irritation, either sharply localized superficial pressure points may develop at ligamentous and aponeurotic attachments, or more diffuse areas of tenderness may develop in muscles and sheaths. This has been well demonstrated by the studies of Pitkin, as well as those of Steindler.

The sensory impressions of pain and tenderness, conveyed to the spinal cord as a result of pathologic lesions in the periphery may, furthermore, be

mediated reflexly through the anterior divisions of the spinal nerves of the same segmental distribution. Such reflex irritation of the anterior divisions of the spinal nerves may induce muscle spasm and postural anomalies identical to those resulting from direct pressure on the anterior nerve roots.

The lumbosacral and sacro iliac joints, for example, are supplied by the posterior nerve roots of the sacral plexus, and pain is referred from these joints only to a small dorsal area over the sacrum. From the ligaments around these joints, however, pain is referred by the anterior nerve roots to large areas in the leg, from the iliolumbar ligaments through the first, second and third lumbar nerve roots, and from the anterior sacro iliac ligaments through the second and third lumbar roots to the front of the thigh and knee, and a small area on the back of the thigh, and from the posterior sacro iliac ligaments through the fifth lumbar, and the first, second and third sacral roots to the back of the thigh and leg, the outside of the leg, the sole of the foot and the big toe (Kersley). It is obvious then that strains or sprains of ligaments and muscular periarthral fibrosis (tendinitis) about the lumbosacral and sacro iliac joints may give rise to diffuse pains along the course of distribution of various peripheral radicles of the lumbosacral plexus. These pains along the leg may not be distinctly segmental in distribution. In this way, they supply some hint of their origin, indicating that they are produced by some mechanism other than direct pressure on nerve roots.

The Swedish clinician Lindstedt, contended without qualification that direct pressure on nerve roots is not a cause of low backache or sciatic pain. He maintained that in most cases the pathogenesis of the condition is best explained by overfatigue or strain of the muscles employed in the act of walking. He pointed out the well known fact that static disturbances, such as flat feet or strain and overfatigue in the muscles, whether in the leg, thigh, pelvis or back, influence the lumbar musculature adversely, as the latter enters importantly in the function of locomotion. He therefore regarded sciatic pain as the result of a myalgic process rather than as a primary affection of the sciatic nerve and considered 'muscular overfatigue' as the localized causal factor in the majority of cases. He explained even the definite neuritic changes, such as absence of Achilles reflexes, sensory disturbances in the leg and so forth, as secondary to the myalgic syndrome or as a concomitant of it.

Although the effects of postural strain unquestionably enter into the production of low back and sciatic pain, neither muscular overfatigue, nor any other etiologic factor, may be singled out as the sole cause of all these cases. The obvious and now well established factor of direct pressure on spinal nerve roots by lesions of the articular facets, by protruded intervertebral disks and by other pathologic conditions to be discussed later

indicates that, besides peripheral muscular strain, other pathogenetic factors must also be assigned as the basis for sciatic pain. Indeed many causative factors, operating in a variety of ways enter into the etiology of sciatic pain. The views of Lindstedt, if applied circumspectly, may serve well, however, to focus attention on one important etiologic mechanism—a simple, logical mechanism so ubiquitous and so obvious that it is too frequently ignored.

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CHAPTER XXXIV

SOME MEASURES OF TREATMENT FOR LOW BACK AND SCIATIC PAIN

As experience indicates, no one remedy exists which can be relied upon for cure of low back or sciatic pain, a combination of measures, properly selected and combined, must ultimately be chosen. Obviously, the means to be selected and their arrangement in the therapeutic scheme depends upon the basic causative lesion and its contributory factors. There are, however, certain measures of treatment which have proved their value in many conditions and are therefore frequently employed. For this reason, we shall discuss briefly the special indications for their use, the principles involved, and when necessary the technique of their administration and their probable effect.

RLST

Rest is undoubtedly the most important single measure of therapy one can employ in the treatment of low back or sciatic pain. It is, of course, useless in a condition caused, let us say, by a neoplasm or protruded intervertebral disk which is producing constant pressure on nerve roots. At times, however, even the symptoms from pressure of a protruded disk may abate with rest in the positions which facilitate return of the herniation into the intervertebral space or which cause recession of edema about the root by eliminating the effect of trauma. In practically every other condition responsible for low back or sciatic pain, rest is of tremendous therapeutic value. It is of the greatest help when the underlying pathologic process is of an irritative or inflammatory nature. Local fixation of the affected area, by casts, braces, or strapping with adhesive plaster, affords some relief, but hardly that attained by complete rest in bed. In the latter case, the body as a whole is at rest, the effect of gravity on a weakened area in the lower back is eliminated, and the contributing influence of poor posture in the trunk and limbs is temporarily suspended. Of tremendous importance, also, is the opportunity that is thus afforded for recovery from general fatigue—a most important element in many cases.

To prevent sagging of the bed, one or two hair mattresses are preferable.

to inner coil spring mattresses. A panel of boards between the mattress and spring eliminates the sagging effect of the springs. Not all patients are comfortable on a hard bed; however, some complain of more discomfort and pain when the back is flat. If this is the case, propping the patient up in bed for a few days by means of a back rest may bring relief and he may subsequently be able to lie flat without pain. Sometimes the mattress must be changed or the boards removed to afford relief from discomfort.

Flexing the knees and supporting them by pillows aids by relieving spasm and tension of the psoas and hamstring muscles. A lumbar pad is frequently useful in maintaining or restoring the normal lordosis. When there is much spasm of the paraspinal muscles, the use of the pad may be intolerably painful. In that case, it may be inserted for only a few hours at a time until muscle spasm has subsided.

FIXATION OF THE BACK

If the patient is in bed, local fixation of the back may be unnecessary, though during the most acute phases of the process some form of fixation is usually temporarily useful. Any form of removable support is to be preferred since it permits the application of baking and light massage. Strapping with adhesive plaster, if properly carried out, may be effective, but removable plaster casts or a posterior plaster shell are preferable.

Fiske has employed successfully a posterior plaster shell and meticulous nursing technique whereby absolute rest is maintained during the entire period of recumbency. In addition to controlling the position of the spine when the patient is in supine recumbency, the shell acts as a splint for the spine during moving and handling of the patient. In a series of cases of chronic backache caused by trauma, chronic arthritis, or both, Fiske found the average duration of treatment with the shell technique plus physiotherapy to be fifty-two days. On discharge from the hospital, 82 per cent of all patients were entirely symptomless; the others had mild symptoms. Follow-up results (after one year) revealed 70 per cent of the patients to be free of symptoms.

The following is a résumé of Fiske's description of the technique of preparation of the plaster shell and the nursing care of the patient.

The patient is covered with stockinette shirting extending from the shoulders to the fold of the buttocks. He lies prone on a hard mattress or table with a small pillow under the abdomen to prevent increasing the lumbar lordosis or to partially correct any lordosis present. This is important as the corresponding elevation in the shell will give discomfort when the patient is turned on his back. Wide plaster bandages, preferably 8 inch, are then passed across the back to an assistant standing opposite who catches the edges of the bandage

with his index fingers, so that it is folded at a point slightly anterior to the mid lateral body line. The operator also folds the bandage on his side at a corresponding point. The cast is gradually built up from a level with the fold of the buttocks to the spines of the scapulae, being somewhat narrower from the axilla upward. A thickness of $\frac{1}{4}$ to $\frac{1}{2}$ inch, depending on the size and weight of the patient, is sufficient. A separate linen pad, filled with cotton and well quilted, is used between the shell and patient. This pad is about 1 inch thick, and large enough to extend 2 to 4 inches beyond all edges of the shell. Two pads are furnished each patient, to permit drying when necessary.

The patient lies on his back in the shell, with a pillow under his head and one under his knees. A draw sheet, extending from the axilla to the hips, is placed under the shell. The patient is allowed to move his legs and arms to the extent that this motion does not strain or move his back. He must not reach, lift or twist at any time and must be entirely limp when being moved by the nurses.

To bathe or turn the patient for any purpose, the pillows are removed, and he is lifted on the draw sheet toward one side of the bed, a nurse lifting each end of the draw sheet. The arm on the side toward which he turns is laid at his side and a small pillow is placed on his abdomen. He is then rolled over in the sheet onto his abdomen, this movement being at an even speed, to prevent lagging or jolting. The shell and pad are then removed. To return patient, each step is reversed.

To prevent turning or using the arms, the patient is fed by a nurse until he has been sufficiently raised on the bed rest to use a bed table in front without lifting his shoulders from the shell.

To use the bed pan, the lower end of the shell is lifted by one nurse, while a second nurse places the upper end of the pan, preferably of the fracture type, under the shell (never between the shell and the patient). The back rest is then elevated sufficiently to bring the shoulders above the hips. When taken off the pan, the back rest is lowered first, and the pan is then removed.

The technique of getting the patient out of bed, when he begins to sit in a chair, must be carried out very carefully to avoid bending or twisting the spine, or any strain due to muscular effort.

In most cases some form of support for the lower back and pelvis is essential for local (protective) fixation of the affected area when the patient becomes ambulatory, after he has been relieved by recumbency. The objective is, of course, prevention of irritation from motion, maintenance of normal postural alignment, and avoidance of strain from unguarded motion and excessive physical activity. The belt or brace must be so designed as to properly immobilize the affected area, it must not do less and it need not be unduly cumbersome. The support must be accurately and carefully fitted. For men, a canvas belt may be employed, for women a brace may be used or, what seems preferable to most of them, a well fitted,

inexpensive cloth corset, lacing in front. A lumbar pad with steel bars is inserted in the back of the support and lateral steel stays may be added for those who require more complete fixation. The belt or corset may have to extend quite high up the back in order to afford sufficient support along the full length of that portion of the lumbar spine affected by the pathologic process. When a support is inadequate for the purpose or is so poorly fitted as to cause discomfort, it is only a burden.

PHYSIOTHERAPY

The principles of physiotherapy in rheumatic conditions have been already discussed (page 231). With few exceptions, those principles apply also to the treatment of low back conditions. Baking or diathermy and massage are to be administered daily. When heat affords relief, and the patient tolerates it otherwise, it may be employed for relatively long periods—even for an hour or two each day—administered in several sessions. If diathermy increases the pain, as it does in some cases, it must be discontinued, however, such patients may tolerate radiant heat well, with relief from pain.

Postural exercises are of great value in most of these cases, for protection of the involved area must eventually devolve upon natural muscular support. Normal muscle balance must be attained to replace the artificial external support, which may then be gradually withdrawn. Postural exercises are particularly necessary when muscular atrophy has occurred after long standing sciatic pain, when abdominal and back muscles lack postural balance, or when the feet are weakened by flaccid, static abnormalities. Obviously, postural rehabilitation must be deferred until the acute, active stages of the process have passed and muscle spasm has disappeared entirely. Even in chronic low back conditions in which postural strain has been solely responsible for the disability, and in which muscle spasm may be absent, it is generally well to allow a lapse of time for recovery from general fatigue before beginning postural exercises. Increasing the burden of general fatigue by exercises, no matter how necessary they may be for eventual recovery, is like flogging a tired horse. Both are ineffective.

EPIDURAL INJECTION

The injection of solutions into the sacral epidural space (through the sacral hiatus) is occasionally useful in relieving severe sciatic pain (Sicard, Evans, Ott, Craig and Ghormley). Various solutions have been employed: procaine hydrochloride, physiologic saline solutions, and 40 per cent anti-

pyrime solution Procaine hydrochloride in 1 or 2 per cent solution appears to be most effective. Relatively large quantities (40 to 60 cc) of the solution are required. It is suspected that one means by which the benefit of

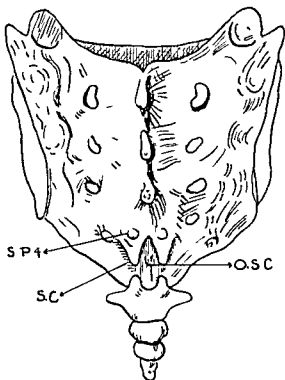


FIG. 115. Posterior view of sacrum and coccyx showing the anatomical boundaries of the outlet of the sacral canal (OSC). SP4 spinous process of the fourth sacral vertebra. SC sacral cornu. (After Evans.)

epidural injection is secured is through mechanical separation of adhesions about nerve roots, another possible means is the analgesic effect of procaine. Single or multiple injections may be necessary. Craig and Ghormley obtained their most satisfactory results from injection of 40 to 60 cc of a 1 per cent solution in typical cases, and a like amount of a 2 per cent solution in more stubborn cases.

During the injection the sciatic pain may increase, to be followed by relief shortly afterward. If only partial relief of pain is obtained, the epidural injections may be repeated at intervals of one or two days. Craig and Ghormley found that 50 per cent of their cases obtained minimum relief after one injection, 16 per cent, were not relieved until two injections were given, 8 per cent, required three or more before relief was obtained.

Reactions of varying degrees of severity, with complaints of fainting, pal

itation, and vertigo, occur not infrequently. They resemble the usual "procaine reactions." No serious undesirable reactions are likely to occur, but if the reaction should become severe, it is safer to stop the injection.

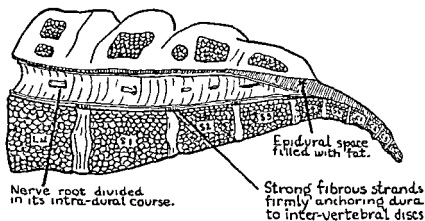


FIG. 116. Sagittal section of lumbosacral region showing the anatomical relationships of the sacral epidural space. (After Evans.)

Evans describes the technique of injection as follows:

The patient lies on the affected side with thighs and legs slightly flexed. The triangular outlet of the sacral canal is then defined in the following way: The tip of the coccyx is first located, and the palpating finger is made to slide upwards toward the sacrum for a distance of about 2 inches. At this point on either side of the midline the prominent cornua of the sacrum can be readily felt in most cases. Above the cornua the imperfect laminae of the fifth sacral vertebra are found to converge to form the apex of the triangular outlet of the sacral canal. The mid point between the two sacral cornua marks the site of the injection (Fig. 115), and an arrow pointing to this spot should be outlined on the skin by means of a grease pencil. The skin and the track of the needle is then infiltrated with 2 per cent novocain, and sufficient time is allowed for the production of efficient local anaesthesia. A lumbar puncture needle is then introduced into the sacral canal (Fig. 116). A syringe is applied to the needle, and an attempt is made to withdraw the piston for a short distance. If cerebrospinal fluid is aspirated the needle has been advanced too far and must be withdrawn a short distance. This precaution is necessary in order to ensure that the point of the needle lies in the sacral canal outside the spinal theca. The sterile solution, previously warmed to body temperature, is then introduced slowly by means of a 25 cc. syringe. The operation throughout must be conducted under aseptic precautions. The patient should rest quietly on the affected side for half an hour, and remain recumbent for 24 hours after the injection.

Epidural injection is a useful therapeutic procedure, frequently bringing about complete or marked relief from pain. Its usefulness is limited, however, to purely symptomatic relief; it does not insure against recurrence.

Its primary function is to relieve the acute pain before something further can be done that may ensure more lasting results. Though it may not be employed in lieu of a diagnosis, epidural injection may bring out valuable diagnostic data. At times these may suggest the possible presence of an intraspinal lesion encroaching upon the domain of the caudal nerve roots or producing obstruction of the caudal sac. However, the diagnostic data available through epidural injection will be discussed in detail later when we describe Love's "reversed Queckenstedt test," employed in the study of patients with suspected protrusion of lumbar intervertebral disks (page 503).

PARAVERTEBRAL INJECTIONS

Paravertebral injection of procaine solution (Steinbrocker), alcohol, or alcohol and neocaine (Labat & Greene) has also been recommended for relief of intractable sciatic pain. Complications may follow such injections, particularly when alcohol is employed, and permanent damage to roots of the cauda equina may occur, leading to paresis of the bladder and bowel.

INJECTION OF THE SCIATIC NERVE

Injection of the sciatic nerve with alcohol, saline solution, or other substances is in our opinion, not warranted, because of the danger of producing permanent damage to the nerve, with paralysis and anesthesia.

REMOVAL OF FOCAL INFECTION

Eradication of definite foci of infection may be distinctly beneficial in certain cases of low back or sciatic pain, not in many. Foci of infection have been removed too indiscriminately in the past, with inevitable disappointment. Before removal is attempted, a diagnosis of an inflammatory basis for the condition—either primary or contributory in character—*should have been established. There should be some tenable direct or indirect evidence implicating an existing focus of infection.* Obviously, the mere presence of tonsils (even if they appear infected), the existence of prostatitis or the indication of other focal infection, does not imply that these are in any way related to the low back or sciatic pain. Promiscuous eradication of focal infection in the treatment of the conditions under consideration has been not only disappointing but disgruntling to patients who had been subjected to such operations, only to be subsequently relieved by simple measures which had an etiologic bearing on the condition.

ROENTGENOTHERAPY

Roentgenotherapy has its advocates (Sicard, Ford, Kahlmeter). It is of some value in inflammatory conditions, although it has been suggested for use in hypertrophic arthritis also. Roentgenotherapy, it must be remembered, is again but a form of symptomatic treatment. Its effect is apparently induced through the analgesic properties of irradiation and the possible destructive effect of x rays on cells of the inflammatory exudate. The subject has already been discussed in some detail (page 242).

INTRAVENOUS INJECTION OF FOREIGN PROTEIN

The intravenous injection of typhoid vaccine for its "foreign protein effect," with induction of fever, has its place, perhaps, in cases of "lumbago," myositis, and inflammatory processes about the intervertebral foramina, lumbar spine, or sacro iliac joints. These are the conditions in which focal infection may play an etiologic role. Treatment with foreign protein in such cases, if employed at all, is logically indicated subsequent to eradication of focal sepsis.

MANIPULATION

The manipulative treatment of low back and sciatic pain has a background distinguished by both curative and harm provoking results. It has been successfully employed for years. However, the damage it may cause in some cases cannot be overlooked. Many of us have encountered patients suffering more serious damage and disability from such manipulation than from their previous disease.

Injury to the cauda equina and temporary or permanent paralysis of the limbs and of the function of the bladder and bowel may result. Such disastrous effects are most likely to develop in patients who have some intraspinal lesion (a protruded intervertebral disk, neoplasm, or thickened ligamentum flavum) which has been overlooked as the etiologic factor. With care in establishing the diagnosis before instituting treatment, such conditions may be readily discovered. And by excluding this group of patients from manipulative treatment the dangers of the procedure are considerably minimized.

The presumed purpose of manipulation is to restore the normal range of spinal movements by breaking down adhesions resulting from sprains, by stretching spastic or contracted muscles, and by unlocking and obliterating

slips at the sacro-iliac joint. It has never been satisfactorily proved how ever that these things are actually accomplished. The value of the procedure must therefore be judged empirically. Manipulation may be tried when chronic back strains (lumbosacral or sacro-iliac) exist or when slips at the sacro-iliac joint can be demonstrated particularly if there is association of the latter with spasm of back muscles hamstrings or both. Riches in England has reported successful results in about 90 per cent of the cases of sacro-iliac and chronic back strain treated by manipulation. He adds however that it must be followed by efficient after treatment. Pitkin Gray Gilcreest and others in America have also urged manipulation as an effective measure of treatment of low back pain attributable to lumbosacral and sacro-iliac strains and sprains.

Manipulation may be performed under anesthesia nitrous oxide supplemented by ether may be employed to obtain maximum relaxation. For those experienced in its use pentothal administered intravenously is an ideal anesthetic for this purpose. The actual steps of the manipulative procedure vary with different operators. The method most commonly employed is the *Baer maneuver* essentially flexion of the extended leg on the body to the fullest range possible in the given case. Sometimes it is possible to bring the toes of the flexed leg almost to the opposite side of the patient's face frequently only to an angle of 50 degrees. In cases with long standing contractures and possibly adhesions the maximum degree of flexion may be attained only in two or three stages.

Others add to the above procedure forcible flexion of the spine rotatory movements of the pelvis to one side and the other and forcible hyperextension and lateral flexion of the spinal column.

Some prefer manipulation without anesthesia when possible. Complete voluntary muscular relaxation is an essential requisite and not always easily secured. The procedure most frequently employed is described by Gilcreest essentially as follows.

With the patient lying on his back relaxed to a maximum degree the manipulator grasps in each hand the patient's heel and ankle rocking the lower limbs on the pelvis slowly and gently with a rotatory motion. The *Gaenslen maneuver* is then performed. The patient fixes the pelvis by flexing one leg completely at the knee and hip with the other extended and hanging over the edge of the table. The manipulator then exerts pressure on the extended leg (in hyperextension) while counterpressure is exerted on the one which is flexed. The hyperextension maneuver is then repeated on the opposite leg.

For reducing sacro-iliac slips and for releasing a sacro-iliac joint presumably locked at extremes of its normal range of motion the so-called

Pitkin maneuver is carried out Gilcreest describes this manipulative procedure as follows

The patient is put on his side with the lower extremity nearest to the table extended fully and the other one flexed at the knee. Pressure is exerted downwards on the flexed knee while counterpressure is put on the shoulder of the same side so as to obtain a rotation of the spine, clockwise of the pelvis and counterclockwise of the shoulders. This same maneuver is repeated with the patient on the other side, changing the position of the legs. During this maneuver, which must also be carried out gently but firmly a definite snap is frequently felt by the patient and may even be audible to bystanders. When this occurs the pain usually ceases instantly, the patient is completely relieved or may feel only a soreness in the low back. The movements of the back immediately become freer, and the patient delights in trying them out, and as soon as he gets off the table will, almost invariably, bend forward, a movement which he could not make previously, to see if he can reach to his shoes.

OPERATIVE TREATMENT

Although conservative measures of treatment will adequately relieve or cure most individuals suffering from low back pain, operative treatment is indispensable in a small proportion of cases. The final decision as to the necessity for operation must of course be left to the judgment of the orthopedic surgeon, nevertheless, just when such aid should be enlisted must be clear to the physician.

We are not concerned here with the technical phases of the various operations that have been devised and suggested. For those seeking such details we have included bibliographic references to comprehensive papers describing the surgical technique of various operative procedures. We are concerned here with a statement of the principles which should govern the selection of patients for operative treatment. For no factor is more important in determining the success of surgical treatment of low back conditions. It is, of course, as unreasonable to deny a patient the benefit of relief from operative treatment when he needs it, as it is to subject him to it when it is ill advised.

Obviously, the proper selection of patients for operation, as for any other treatment is impossible without an accurate diagnosis. There are conditions in which operative treatment is indicated as soon as the diagnosis is established. Among such conditions are protrusions of intervertebral disks, thickened ligamenta flava, operable intraspinal neoplasms, some cases of spondylolisthesis and probably most cases of tuberculous arthritis. These conditions will be discussed in detail later. We cannot urge too forcefully

the advisability of careful neurologic study for the exclusion of intraspinal lesions before arthrodesing operations on the lower spine are performed

There are on the other hand conditions causing low back pain in which operative treatment is to be largely avoided. As a rule, conservative treatment of chronic arthritis of the spine or sacro-iliac joints is more justifiable than is surgical. Such practice is applicable to both hypertrophic and atrophic types of arthritis. There are some exceptions to this rule such as advanced but still painful sacro-iliac or lumbosacral lesions in which the inevitable but long delayed natural ankylosis should be anticipated. We will refer to this phase of the subject more specifically in a discussion on spinal arthritis (page 459). Medical treatment will effect spontaneous fusion in most of these cases eventually producing either arrest of the disease or relief from symptoms. Another reason for avoiding operation for such conditions is their tendency to progress beyond the confines of the circumscribed arthritic involvement for which fusion may at first seem advisable.

Patients with traumatic neuroses and other functional nervous states will obviously not be relieved by operations on the spine. Nor will patients with malignant lesions or constitutional affections benefit from surgical arthrodesis. Those with low back disability who are engaged in legal maneuvers to obtain financial settlement are notoriously poor subjects for successful operative management. Even when the condition unquestionably demands it its success is more likely to be assured by waiting until a satisfactory financial settlement has been effected. Surgical treatment is likewise out of the question for aged patients or for those who are poor surgical risks.

The remaining patients in whom surgical operations are sometimes indicated are those with localized traumatic hypertrophic arthritis affecting either the bodies of the vertebrae or their articular facets and those with recurrent back pains caused by repeated strains and sprains which are induced largely by occupational stress and are relieved by rest. In the latter case the object is to provide permanent fixation of the back by surgical arthrodesis. The indications for fusion in certain conditions of the sacro-iliac joints will be discussed later (page 476).

Certain specific operative procedures are indicated in certain types of cases for example laminectomy for excision of protruded disks thickened ligamenta flava or intraspinal tumors. Operations on the iliotibial band (Ober fasciotomy) and Freiberg's myotomy of the piriformis muscle will be discussed later. The author has had no personal experience with Heyman's procedure of subperiosteal stripping of the gluteus maximus muscle and has encountered no cases in which it has been employed. Arthrodesing operations are the ones generally required in the surgical treatment of most other low back conditions. The aim of the operation is of course to sta-

bilize the involved joint permanently, to free it from irritation, and thus relieve it from pain. In lesions of the articular facets (causing pressure on nerve roots), decompression of the intervertebral canal by facetectomy may be demanded.

Obviously, the site of the lesion responsible for the pain must be well localized either to one or both sacro iliac joints, or to the lower lumbar or lumbosacral area. As Compere says, "The operation of transsacral fusion is in the nature of a shot gun procedure and should rarely be advised."

When all the facts are considered, the indications for surgical arthrodesis for low back pain are rather few and the employment of operative treatment is called for infrequently. Nevertheless, it is a phase of treatment fraught with potentialities for much good or harm.

Operation should be decided upon and performed by one with experience. As Compere says: "The judgment and skill of the orthopedic surgeon may be measured by his ability to select, from the great numbers of patients who consult him because of symptoms of low back pain, those with real indications for operative treatment. His courage may be shown either in refusing to perform an operation when it is urged that he do so and his conscience and judgment say 'no,' or in his determination to operate when he is convinced that only by such a procedure can the patient be cured." He points out that of 2,242 patients who came to the University of Chicago clinics seeking relief from low back pain, only 76 (3.4 per cent) were subjected to operative treatment. This indicates an attitude of conservatism which is, fortunately, quite generally practiced by those with extensive experience.

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CHAPTER XXXV

THE CAUSES OF LOW BACK AND SCIATIC PAIN

CLASSIFICATION

A classification of all the factors that may cause low back or sciatic pain would make up a long list of extremely diverse pathologic conditions. Aiming for a practical rather than an encyclopedic discussion, we have limited our consideration to those conditions most likely to be encountered by the general practitioner

- I Strains and sprains
- II Postural strain
- III Arthritis
- IV Fibrositis (myofascitis)
- V Congenital anomalies
- VI Abnormalities at the sacro-iliac joints
- VII Affections of the piriformis muscle
- VIII Contracture of the iliotibial band
- IX Abnormalities of the vertebral articular facets
- X Lesions of the lumbar intervertebral disks
- XI Thickened ligamenta flava
- XII Tumors—rheumatism requiring morphine
- XIII Miscellaneous
 - I fractures
 - Dorsolumbar sprains
 - Pressure from lesions in the pelvis and rectum
 - Constitutional diseases
 - Toxic factors
 - Primary (idiopathic) sciatic neuritis and herpes zoster
 - Vascular diseases
 - Functional nervous states
 - Malingering

These topics will be discussed in the chapters that follow

CHAPTER XXXVI

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

I MUSCULAR AND LIGAMENTOUS STRAINS AND SPRAINS

Strains of the back are probably the most common single cause of low back disability encountered in office and in clinic practice. Lumbosacral strains are far more frequent than sacro iliac. They may develop suddenly and acutely, particularly among those engaged in strenuous occupations. The back may be strained during a fall, it may be wrenched slipping or while lifting a heavy object, or during any physical activity which involves motion at the lower back. Even the most trivial activity, such as stooping to lace a shoe may precipitate a strain of the back, nor does the degree of trauma necessarily determine the severity of the symptoms.

The pain may be agonizing from the start, so that the patient cannot straighten out when he stoops and must be helped to bed. In most cases however, the pain at the onset is not so severe. Perhaps it is only a "stitch" in the back, which abates within the next hour or two. It recurs later, however, as a dull ache, aggravated by motion, and gradually increasing in severity as the patient continues at his customary activity. The pain may never become totally disabling, on the other hand, it may gradually increase to such proportions as to incapacitate the individual and force him to bed.

In milder cases the pain may be confined to the back. When more severe, it may affect a large area of the lower lumbar region, and be most acute over one side of the lumbosacral or sacro iliac area. In such cases, the pain frequently extends into the thigh and leg. With lumbosacral involvement the radiation is most likely to be along the posterolateral aspect of the thigh and leg, to the external malleolus and dorsum of the foot. With sacro iliac involvement the pain is more likely to extend to the buttocks, the posterior aspect of the thigh, and the adductor regions. However, these modes of pain distribution are not diagnostic.

Following several attacks of low back and sciatic pain, the pain in the leg may persist after the backache has entirely disappeared. In these cases, the patient is more than likely predisposed to frequent strains because of

some underlying chronic pathologic process in the intervertebral joints with severe root irritation. Such strains of course, clear up more readily than the effects of the more chronic joint lesion.

When acute the pain in the back is generally of a sharp lancinating type; there is also a burning sensation along the thigh and leg when referred pain coexists. The backache is aggravated by motion and largely relieved by rest, although attempts to turn in bed may cause discomfort. In bed the patient is likely to assume the position of maximum relaxation that is with the body, hips and knees moderately flexed and the knees supported by pillows.

The objective findings depend on the severity of the condition and on the site chiefly affected. The patient may stand with the body bent forward, unable to straighten because of pain. There is generally a list to one side—sciatic scoliosis—directed either toward or away from the affected side. Usually there is marked muscle spasm. With lumbosacral involvement the normal lumbar lordosis may be entirely obliterated. The flat rigid low back, which does not permit motion in any direction because of pain, is characteristic of the more severe cases. Tenderness may be elicited in such cases on pressure over the affected area and with lumbosacral lesions, not infrequently to a lesser extent on the opposite side, too. Tenderness may also be elicited over the gluteal muscles and posterior aspect of the thigh. Straight leg raising (Lasegue sign), flexion and hyperextension of the hip are limited and cause acute pain.

In milder cases all of these signs are less pronounced. A limited range of motion may be possible, but it causes pain both over the affected region in the back and along the leg, corresponding to the area of distribution of referred pain. When the strain is confined to the sacro-iliac region, forward bending may be more free, as the patient sits with the hamstrings relaxed. The Lasegue sign is most likely to be confined to the affected side in sacro-iliac lesions; it is bilateral though more pronounced on the affected side in lumbosacral strains. Compression of the ilia with the patient lying on his side and pressing the spines of the ilia downward and outward, as he lies in the supine position, may elicit pain in sacro-iliac strains but causes no discomfort with lumbosacral involvement.

PATHOLOGY

It is assumed that sprains of the back represent actual tears of the muscles, ligaments or joint capsule and that strains do not. Actually it is difficult to differentiate clinically those cases in which ligamentous tears have occurred and those in which they have not. It is entirely possible that all strains of traumatic origin which are sudden in onset have as their basis

actual rupture of some fibers of the ligaments or joint capsule. It is wiser to assume that it is so in the cases showing severe low back disability and to treat the patient accordingly from the start. In this way he is given the chance to forestall serious chronic disability which is so likely to follow inadequate treatment.

As the work of Steindler indicates, the referred pain in some cases probably results from irritation of branches of the posterior divisions of the spinal nerves supplying the affected ligaments, with reflex effects mediated through the anterior divisions of the spinal nerves of the same segmental distribution. In other cases, particularly with low lumbar or lumbosacral lesions, the referred pains may be the result of direct irritation of the fourth and fifth lumbar nerve roots by an exudate or synovitis of the corresponding intervertebral joints.

Sprains of the back may be associated with arthritic changes in the spine, or congenital anomalies, or pre-existing postural strain of long duration, all of which factors increase greatly the susceptibility of the back to sprains from causes which otherwise would produce little damage. When the trauma has been severe, the sprain may be associated with a fracture at the point of ligamentous attachment to the bone.

Röntgenographic examination is important in establishing the existence of arthritis, lesions of the articular facets, fractures, congenital anomalies, and the like. The discovery of such associated lesions not only clarifies the prognosis, but may alter the procedure in treatment.

TREATMENT

Proper treatment instituted immediately after the onset of the condition is most likely to prevent recurrent, chronic disability which is otherwise likely to develop.

If the symptoms are at all pronounced, rest in bed should be insisted upon. In the more severe, acute cases rest is not only imperative, but generally welcome to the patient. The bed should be made firm by means of a fracture board inserted between the mattress and spring. A position of muscular relaxation with the knees flexed and supported by pillows, affords some relief from pain. In the acute stages the lumbar spine should be maintained in the position of maximum comfort for the particular patient. Some patients feel better with the lower back in a position of slight flexion, others are more comfortable with support by a lumbar pad.

Heat applied to the back, either by baking or hot fomentations once or twice a day, and light massage are distinctly helpful. For this reason immobilization with a plaster cast which precludes the employment of heat is

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Heat applied to the back either by baking or hot fomentations once or twice a day, and light massage are distinctly helpful. For this reason immobilization with a plaster cast which precludes the employment of heat is

generally inadvisable. When necessary, fixation of the spine by firm strapping with adhesive plaster is sufficient.

In acute stages no attempt should be made to correct forcibly the abnormal postural attitudes encountered. They are the result of muscle spasm and constitute a temporary protective measure against pain and further irritation. As the muscle spasm subsides through prolonged rest, the scoliosis and other acute postural defects disappear.

To aid the resolution of muscle spasm, when it is marked, and when it does not yield to rest and immobilization alone, gradual head and leg traction with weights and adhesive plaster strapping may be indicated. Pain and muscle spasm should not be increased thereby, if they are, the weights should be reduced or traction entirely discontinued.

Analgesics codeine when necessary, and sedatives are generally useful during the most acute phase of the process.

Rest in bed may be required for only a few days in the mild cases, or for weeks or for two or three months, in more severe or recurrent cases with a history of previous attacks which were inadequately treated. When the patient becomes ambulatory he is fitted with the proper type of support designed to maintain immobilization of the affected region of the spine. For lumbosacral lesions a wide lumbosacral belt or brace is required. For women the brace may be incorporated in a front laced corset.

Physiotherapy is continued until the active process has entirely subsided. At that time postural exercises are instituted to correct any existing postural deviations. Static defects in the feet are corrected with proper shoes and supports until physiologic postural correction has been effected.

In the more resistant cases, particularly for those patients who first appear for treatment some time after the onset of the injury, prolonged rest, physiotherapy and traction may be required. Manipulation under anesthesia may be helpful. It is difficult to predict, however, just when manipulation may help and when it may not. Probably the best indication of the need for it is chronic persistent muscle spasm in the back as well as in the hamstrings. It is important to make sure that no lesion exists at the intervertebral foramen or the intervertebral disk before manipulation is attempted lest more harm than good be done.

When chronic disability is due to distinct, localized pathologic changes at the lumbosacral or sacro-iliac articulations which predispose to recurrent exacerbations of low back strain, bony fusion—arthrodesis—of the affected joints is likely to be successful. Before surgical treatment is attempted however, it is well to make sure that the conservative plan of treatment has been perseveringly followed with scrupulous attention to detail. It is only when these measures have been conscientiously tried and found ineffective that surgical arthrodesis is entirely justifiable. The decision as to the

advisability of operative fusion must be entrusted to an experienced, competent orthopedist, preferably to one who is conservative in his attitude toward surgical arthrodesis. When indicated, operative treatment may yield extremely gratifying, permanent relief of recurrent disability. It is understood, of course, that careful diagnostic exclusion has been made of those conditions causing low back pain, to be described later, that demand purely surgical management. In other cases, the attitude of most physicians and orthopedists intimately acquainted with the problem is to turn toward surgical arthrodesis more and more cautiously.

In the consideration of persistent, chronic disability following injury, thought must be given to the possibility of the existence of a traumatic neurosis, which will, of course, not yield to treatment directed to the back.

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CHAPTER XXXVII

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

II POSTURAL STRAIN

Chronic postural strain exerted on the muscular and ligamentous structures of the lower back is among the most common causes of low backache and sciatic pain

The background in almost all such cases is a musculature that is inadequate for the demands upon it. The body form is disturbed by failure of balance, whereupon secondary conditions, both extrinsic and intrinsic develop and complete the vicious circle.

The postural disturbances causing such backache may start with flat feet or pronation, which leads, in turn, to secondary postural strain in all groups of muscles used coordinately in walking and maintenance of posture—the muscles of the legs, gluteal regions, and back. The condition may be induced or aggravated by many factors such as corpulence, occupational overexertion, or by disease processes which cause pain and protective over-tension in the muscles of the legs or back. Kerr discussed in great detail and illustrated clearly the effect of obesity with a sagging or protuberant abdomen, which by exerting an excessive downward pull leads to muscular overfatigue, strain on the ligaments supporting the lumbar spine, and backache. An occupational postural strain induced by standing for long hours in a stooping position, is exemplified best by dentists and surgeons who are not infrequently subject to such low back disabilities. The postural strain may originate in any part of the lower limbs or back and may be secondary to congenital or acquired disease, or to deformity in the bony, muscular, or ligamentous structures.

Regardless of the nature of the primary postural defect, its effect, so far as the production of low backache and sciatic pain is concerned, is muscular overfatigue. The latter may aggravate the initial postural disturbance, producing strain on ligaments all about the lower spine and sacro iliac joints.

The resulting manifestations at first barely perceptible gradually become more disturbing and intense. Pain may originate directly in the muscles or ligaments involved, particularly in those around the articulations of the

lower back. But if the process is of long standing and neglected pain may be referable to mechanical strain at the joints of the lower spine particularly at the intervertebral joints where pressure exerted on nerve roots may produce typical sciatic pain. Hypertrophic arthritic changes at the intervertebral joints perhaps induced by postural trauma increase the likelihood of nerve root involvement and thereby the degree of disability.

Generally the symptoms appear insidiously and without cause so far as the patient can tell. The discomfort is generally inconstant appearing intermittently in varying degrees of severity.

Many systemic factors—such as anemia, inanition or obesity, general body overfatigue from excessive physical exertion, nervous or mental strain and debility induced by acute illness or chronic infection—are especially prone to bring on disability. We are excluding for the moment the effects of muscular strain from acute trauma which may light up the symptoms of chronic postural strain hitherto barely perceptible.

Such patients complain rather frequently of a dull ache or a tired feeling in the lower back, seldom of severe pain. As a rule the discomfort is confined to the low back and it is frequently bilateral. It may be most marked directly over the lumbosacral junction or over the sacro iliac region depending on the point of maximum stress. The symptoms may be most pronounced toward the end of the day, especially after a hard day's work or if the patient gets tired. In that case he may be relieved by lying down or by resting. A tired feeling or dull pain may be projected to the gluteal regions, the thighs, the back of the knees or the calves; in more severe cases typical sciatic pain may occur.

Muscle spasm is discernible only in the severe, more acute cases. Without exception various types of postural abnormalities are encountered. They may not be glaring and may indeed be easily missed in some cases if the examiner does not look for them deliberately. In others the posture is obviously poor. The patient stands with head forward, shoulders drooped, chest flat, abdomen protruding and the lumbar spine in marked hyperextension. The feet are pronated and the body weight rests largely upon the heels. Too many of such gross postural defects are taken for granted and hardly considered in relation to the low back disability. Disregarding the obvious relationship between such gross postural defects and the backache, the physician too often unwittingly focuses his attention on the patient's tonsils—if they are still present—and orders them removed with inevitable disappointment later.

In most cases some tenderness in the lower back may be elicited on deep pressure, the point of maximum tenderness depending again on the site of maximum strain. Generally there is pain at the lumbosacral region on hyperextension of the spine or hips, but less discomfort on forward bending,

As a rule there is full freedom of all motions at the lower back, except in more severe cases in which muscle spasm may limit somewhat forward backward, or lateral motion

If the strain is largely at the sacro iliac joints or ligaments, lateral compression applied at the anterior superior spines of the ilia or pressing the spine downward and outward, as the patient lies supine may reproduce the pain of which the patient complains. Definite tenderness over the lower portion of the sacro iliac joints, elicited by pressure through the rectum aids in localizing the process to the sacro iliac joints. Otherwise the manifestations of lumbosacral and sacro iliac strain may be quite similar.

Examination of the back may yield entirely normal findings. The diagnosis may then have to rest on the history and the existence of a source of postural strain. Such a diagnosis must be confirmed by a therapeutic test consisting of correction of the suspected postural error with relief from symptoms.

The general medical examination may reveal significant associated conditions which contribute to lowering of the patient's general threshold to fatigue and pain.

Röntgenograms of the spine, which should include at least antero posterior and lateral views and, preferably, oblique views also, are generally negative, but there may be evidence of congenital anomalies, which are not infrequent among this group, or mild secondary arthritic changes. It is important not to be misled by such anomalies which, though perhaps predisposing to postural strain, may cause no trouble otherwise.

TREATMENT

Treatment of this type of low back disability obviously requires correction of the causative postural error. When the symptoms have been severe and in advanced cases, the patient requires a preliminary period of rest in bed, not only to relieve the strain on the lower back itself, but to relieve him of general fatigue which may be the real precipitating factor in the disability. Attention to the general condition of the patient is as important in bringing about relief as correction of the causative postural error. Correction of any factors contributing to general debility—*anemia, under nutrition, nervous exhaustion*—must be carried out. While in bed, the patient may be relieved by a pad supporting the lumbar spine, and a pillow supporting the knees in flexion. When obesity is a factor, a low caloric diet is instituted. Endocrine abnormalities should, of course, be corrected, whether they contribute to the obesity or not. While the obesity is being treated, temporary support of the abdomen by means of an abdominal belt may yield striking amelioration of the symptoms.

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When rest in bed is unnecessary or impossible a properly fitted belt designed to immobilize adequately the affected region of the spine brings temporary relief of symptoms. For women a padded brace may be incorporated in the corset.

Baking and massage of the muscles should be employed. Static abnormalities in the feet must be corrected. Wedging of the heels to compensate for pronation and the use of properly fitted supports for the arches minimize the effect of static foot strain.

But the ultimate aim in the treatment of these patients is restoration of physiologically normal posture. This is accomplished by exercises aimed at restoring normal body mechanics. In older patients in whom postural re-education may for one reason or another be difficult or impossible postural correction through permanent use of supports may be indispensable. Postural rehabilitation is practical for younger patients. But in any case it is a long range program requiring from the patient cooperation based on understanding of the aim of treatment and the means by which it may be accomplished. Success of such a plan requires intelligent and willing cooperation between patient, physician and competent enthusiastic physiotherapist—a state more easily described than reached. But the reward is relief of symptoms, eventual discard of supporting apparatus for the back and feet and insurance against recurrence.

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CHAPTER XXXVIII

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

III ARTHRITIS

IV FIBROSITIS (MYOFASCITIS)

ARTHRITIS

Arthritis in the lumbar or lumbosacral regions constitutes another common cause of low back pain met in general practice. The arthritis may be of the atrophic type, in which case it is generally part of a widespread process or it may be of the hypertrophic type, occurring for the most part, after middle age.

In the former case there is an inflammatory and exudative reaction in the periarticular soft tissues and atrophy of cartilage and bone. Partial destruction of the articular cartilage or some degree of secondary hypertrophic change may occur. In advanced stages ankylosis at the intervertebral joints (articular facets) may take place. The paravertebral ligaments are frequently affected early by the pathologic process, so that they may be involved long before there is any demonstrable roentgenologic evidence of involvement of the spine itself. This type of spinal arthritis has already been discussed (page 113).

Hypertrophic arthritis, with spurring at the margins of the vertebral bodies, is seen often in roentgenograms of the lower spine, but the condition frequently produces no disturbing symptoms. When low back pain results in such cases it is probably produced by strains secondary to the arthritic defect. Hypertrophic arthritic changes in the lower lumbar spine or at the lumbosacral junction with spurring at the margins of these vertebrae, may actually be only the secondary effect of some other, more fundamental cause: chronic degenerative destruction of the related intervertebral disk, long standing postural defects, traumatic sprains with fracturing of the bone at the points of ligamentous attachment, and so on.

Although spurring at the margins of the vertebral bodies resulting from hypertrophic arthritic changes, is the most glaring pathologic defect evident in roentgenograms of the spine, arthritis affecting the margins of bodies of

the vertebrae is not likely to be confined to that area to the exclusion of similar involvement of the articular facets. Not only is the latter involvement a not infrequent accompaniment but probably the most significant in causing pain. The hypertrophic spurs with the associated involvement of the longitudinal ligaments may contribute to the discomfort confined to the back. But the root pains responsible for the associated sciatic pain are largely the result of pathologic changes at the facets or synovitis at the intervertebral joints with secondary irritation of nerve roots as they traverse the intervertebral foramina. The importance of the latter site in the production of sciatic pain will be referred to again later when we discuss the relation of the articular facets to sciatic irritation (page 485).

A spine affected by either atrophic or hypertrophic arthritis is further more particularly vulnerable to strains and sprains because of limitation of motion and deformity that may exist. The acute exacerbations of low back and sciatic pain suffered by patients with hypertrophic arthritis may indeed be largely the effects of such secondary strains and not of the arthritic changes themselves.

There are of course many types of specific arthritis which may affect the lower lumbar and lumbosacral articulations. Tuberculous, pyogenic and many other types of specific infections may be localized in the spine. I have seen localized syphilitic involvement at the lumbosacral joint producing low back pain clinically indistinguishable from other conditions. The diagnosis established roentgenologically was confirmed by serologic findings. Tuberculosis of the spine must not be forgotten as a possible cause of indefinite pains in the lower back (page 337).

The manifestations resulting from these various types of spinal arthritis are similar to those caused by strains of the spine. The severity of the pain and its distribution naturally vary within wide limits depending on many factors. The discomfort may be confined to the lower back or it may be associated with referred pains along the buttock, the thigh or the lower leg. Frequently the referred pains are bilateral owing to more or less symmetrical involvement of both sides of the spine. In other types of disease of the spine the symptoms are more or less intermittent being caused by factors acting intermittently. In spinal arthritis the symptoms are more apt to be constant though of varying degrees of severity. Owing to the associated muscular fibrositis that is so frequent an accompaniment of this disease there is likely to be stiffness and soreness in the back particularly early in the morning, and waning as the day goes on and as the patient limbers up.

Treatment

The treatment of the various types of arthritis has already been discussed in greater detail (pages 1-9 -9). These details need therefore not be repeated here.

The general systemic treatment of the arthritis is of first importance. All factors contributing to the disease must be corrected as well as possible.

In atrophic arthritis of the spine, resolution of the inflammatory process must be aided and deformity of the spine prevented by rest and protection. At first the patient should be kept in bed, later the use of an appropriate brace or belt may give adequate support (see page 118).

In cases of hypertrophic arthritis, relief may be afforded by proper supports, physiotherapy, correct posture, particularly of the feet and reduction of obesity when that factor exists. In addition to affording relief braces and belts protect the back from strains to which it is so vulnerable. When acute exacerbations occur, induced by straining the back they are to be treated like ordinary strains, and later steps are to be taken to treat the underlying arthritis.

Surgical measures occupy a limited place in the treatment of spinal arthritis. Even for tuberculosis of the spine, in which surgical fusion is frequently of recognized benefit, there is still diversity of opinion among orthopedic surgeons as to the relative merits of nonoperative treatment and surgical arthrodesis. In other arthritic conditions of the spine the need for surgical fusion is even less apparent. In atrophic arthritis of the spine the pathologic process tends only too readily toward spontaneous ankylosis. In hypertrophic arthritis, with proliferation at the margins of the bodies of the vertebrae, the tendency is again toward slow, gradual union of marginal osteophytes, which may produce, in time, desired fixation of the spine and relief from pain. An elderly patient, with marked hypertrophic arthritis which is producing pain, is obviously not a fit subject for operative fusion. The middle aged individual with a localized process, who may benefit from fusion if not relieved by more conservative measures, is susceptible to development of similar degenerative changes in other, perhaps adjacent, segments of the spine. These circumstances emphasize the necessity of weighing the facts from every standpoint before deciding on surgical arthrodesis of an arthritic spine.

And yet there are occasional instances in which circumstances point to the advisability of surgical fusion. Traumatic hypertrophic arthritis, for example, induced by factors no longer active, but with residual changes which are causing severe disability, not sufficiently relieved by conservative means may lend itself ideally to cure by fusion of the affected segments. This situation applies particularly to middle aged patients, who are physically active or whose strenuous occupations may bring forth frequent acute exacerbations of low back disability and who may be restored to useful lives, free of pain through absolute fixation of the affected region. In many of these cases, as will be shown in a later discussion there is constant involvement of the articular facets at the intervertebral foramen, and pressure on spinal nerve roots and sciatic pain. Under such circum-

stances fusion of the spine alone may not afford the desired relief. Resection of the affected facets may be necessary in conjunction with arthrodesis. This phase of the subject will receive more detailed consideration later when we discuss the relation of the articular facets to sciatic pain (page 492). In short, surgical treatment in these conditions should be based on understanding of the problem from every angle. Experience is of the greatest value. By and large the wider the experience of the clinician or surgeon the more conservative his approach to surgical arthrodesis is likely to be.

FIBROSITIS (MYOFASCITIS)

The general considerations on the subject of fibrositis have already been covered in a previous section (page 361) to which the reader is referred for details.

In relation to low back and sciatic pain fibrositis is frequently a factor. Muscular as well as periarticular fibrositis may be responsible for low backache and in exceptional instances for sciatic pain as well. The latter is probably a sequela to the secondary disturbances such as postural strain and other reflex effects induced by involvement of the posterior divisions of the lumbar spinal nerves. Steindler substantiated the foregoing explanation of sciatic pain occurring in cases of myofascitis; he believed it to be a reflex manifestation initiated by local irritation of sensory (posterior) branches of these nerves. In many cases of myofascitis he injected 5 to 10 cc of 1 per cent solution of procaine hydrochloride directly into the affected muscular or ligamentous paraspinal structures. In this way he obliterated the irritation of the affected posterior branches of the spinal nerves and was able to suppress both the local tenderness previously elicited and the pain referred to the leg. Likewise the previously positive Lasegue phenomena presumably caused by reflex involvement of the anterior divisions of the spinal nerves of the same segmental distribution disappeared.

By means of pneumography after the injection of air into the fascial spaces Gratz demonstrated adhesions between the lining of the fasciae of *the back and the muscles they cover. He showed that air in the fascial planes* of relatively normal persons is evenly distributed in the fascial planes of patients with arthritis and an associated myofascitis the air has an irregular distribution.

The diagnosis of fibrositis (myofascitis) is the exclusive cause of low back pain or sciatica may not be easy. The existence of the more typical manifestations of fibrositis (as discussed in Chapter XXIV) particularly the association of stiffness with backache and localized tenderness over muscles and ligaments may be helpful.

Treatment

For general principles of treatment of fibrositis, the reader is again referred to a previous discussion of that subject (page 363)

The importance of treating the patient from the general systemic standpoint needs to be re-emphasized

When the symptoms are more or less acute, rest in bed is necessary. General fatigue is an important aggravating factor in fibrositis. The lower the general physical reserve of the patient the greater the need for rest.

When the symptoms are less marked, or chiefly muscular, properly fitted supports are employed as the patient is allowed to resume activity after the period of rest in those cases requiring it.

Physical therapy, such as the application of heat, massage, graduated exercises, including those aiming at the restoration of normal body mechanics, is an important aspect of treatment in these cases.

Manipulation has been employed to break up fascial adhesions, it should be followed by rest, physical therapy, and systemic management. But manipulation, though helpful, has potentialities for causing harm, and the indications for its use are not always clear.

Gratz has freed fascial adhesions surgically, after localizing the site of the fascial change roentgenographically following air insufflation into the fascial spaces. He obtained "satisfactory improvement" in about half of his cases. The patients operated upon were selected from a group who had not responded satisfactorily to any of the more conservative measures previously employed. Not having had any experience with air injection the writer is unable to evaluate its importance in the diagnosis and treatment of myofascitis. It is a subject worth following, but the general adoption of this procedure is probably unnecessary and perhaps not devoid of danger.

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[See also bibliographic references at end of Chapters IX, XIX and XXVII]

CHAPTER XXXIX

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

V CONGENITAL ANOMALIES

It is not surprising that congenital anomalies in the lower spine as seen in roentgenograms should have aroused so much attention among physicians as a cause of low back and sciatic pain. The lumbosacral area is most vulnerable to such anomalies for it represents a transitional area in the phylogenetic development of our species since man has acquired the erect posture. And so we find here many and various abnormalities of anatomic structure. In the past these were considered one of the principal causes of low back pain. It would perhaps be misleading to say that such anomalies are not a predisposing cause of low back disability. But evidence both clinical and anatomic reveals that they are probably the least likely precipitating cause of the conditions in question. Although roentgenographic evidence of such abnormalities is impressively large, disturbing clinical manifestations of their presence are frequently totally lacking. Congenital anomalies of various portions of the lower lumbar spine, particularly those which may cause instability at the lumbosacral joint, may however predispose to secondary strains, arthritic changes, and so on, which may indeed produce low back disability.

A large variety of congenital defects affecting various portions of the vertebrae are found in patients complaining of low back and sciatic pain. Some of those most frequently encountered will be described.

SPINA BIFIDA OCCULTA

Spina bifida occulta is a lack of fusion between the two halves of the neural arch, occurring either in the fifth lumbar or first sacral vertebra. *Spina bifida occulta* itself assumes clinical importance only when it is associated with a significant degree of congenital myelodysplasia. Then it is the cord defect which is actually responsible for the neurologic manifestations, but such neurologic conditions are generally more extensive than in sciatica; they are accompanied frequently by various grades of neuro

genic disturbances affecting the function of the bowel and bladder Spina bifida occulta may constitute a cause for instability in the lumbosacral region thus predisposing the patient to other superimposed affections which may cause low back pain Otherwise it is merely an incidental anatomic peculiarity It is difficult to conceive how such a defect could of itself be the cause of any of the manifestations of low back disability or sciatic pain as has occasionally been supposed

SACRALIZATION OF THE TRANSVERSE PROCESSES OF THE FIFTH LUMBAR VERTEBRA

This is a fairly common congenital anomaly in which one or both transverse processes of the fifth lumbar vertebra are abnormally large, extending toward the base of the sacrum or ilia Such an abnormality may constitute the basis for a weak back that is one more than commonly liable to strains and to damage from even normal physiologic stress

There may be partial lumbarization of the first sacral segment with a narrow intervertebral disk between the first and second sacral vertebrae Or again there may be complete bony fusion between the transverse process of the fifth lumbar vertebra and the sacrum Such complete fusion may logically be expected to produce a more stable joint It hardly furnishes an explanation for irritation of the roots of the sciatic nerve Steindler says

In the question of the long impinging transverse process and of sacralization physicians are today far from acknowledging the so-called Bertolotti syndrome (sacralization sciatica and scoliosis) Although it is a variation of high frequency (1-6 per cent) many doubt its pathogenic significance

HORIZONTAL SACRUM AND DEEPSLAVED FIFTH LUMBAR VERTEBRA

This abnormality may contribute to ligamentous strain or lead to localized traumatic (postural) hypertrophic arthritis with resulting symptoms referable to the back or to roots of the sciatic nerve by causing instability of the lumbosacral joint

SPONDYLOLISTHESIS

Spondylolisthesis a subluxation usually a slipping forward of the lumbar spine on the sacrum affects chiefly the fifth lumbar vertebra in relation to the sacrum It may cause low back disability and sciatic pain This condition is discussed here because in practically all instances of spondylolisthesis there is an underlying congenital defect which predisposes to subluxation of the lumbar vertebra Separation of the neural arch and spina bifida

occur in the large majority of cases. The defect in the neural arch causes loss of support to the articular facets impairing the strength of the lumbosacral joint and resulting in subluxation.

Spondylolisthesis was formerly thought to be a rare condition but is met more frequently nowadays owing to more frequent consideration of its possible presence and particularly owing to more careful roentgenologic study of the low back. Meyerding has recently reported a series of 583 cases observed at the Mayo Clinic.

That trauma is an important precipitating cause of a slipped vertebra is evident from the fact that the congenital defect predisposing the patient to this condition may exist for years without development of spondylolisthesis. Seventy per cent of patients with spondylolisthesis are those who do hard work (housewives, laborers, farmers, etc.). As Meyerding's series has indicated, 70 per cent too are males. A definite history of trauma may be elicited in about half the cases but the relation of the trauma to the onset of the subluxation cannot always be ascertained. Many of these patients have had chronic backache for months or years prior to the injury, the latter only aggravating the previous symptoms. Ten per cent of patients who have definite spondylolisthesis are entirely unaware of it, the condition being found incidentally during examination for other purposes. This fact indicates then the possibility that trauma, either repeated chronic strain or sudden injuries, may initiate symptoms of a pre-existing though silent anatomical defect.

Although forward slipping of the fifth lumbar vertebra on the sacrum is the most common occurrence, forward slipping of the fourth lumbar vertebra on the fifth occurs in over 10 per cent of cases. Very occasionally other lumbar vertebrae are affected or reverse spondylolisthesis may occur in which the involved vertebra is displaced backward instead of forward. For anatomical reasons, chiefly the normal inclination of the superior surface of the sacrum, reverse spondylolisthesis is more likely to affect vertebrae other than the fifth lumbar.

Meyerding has graded the degree of subluxation as determined from examination of lateral roentgenograms as follows. If the fifth lumbar vertebra has slipped forward less than a fourth of the distance across the lumbosacral joint, the spondylolisthesis is graded 1; if it has slipped less than half the distance it is graded 2; if it has slipped less than three fourths of the distance it is graded 3; and if it has slipped more than three fourths of the distance it is graded 4.

Clinical Manifestations

Spondylolisthesis occurs most frequently between the ages of thirty and fifty years, although it has been observed in children and in the aged.

Backache is the chief complaint in over 80 per cent of the cases. Radiation

of pain to the sacro-iliac regions, the hips, and the legs occurs not infrequently. Some patients complain of pain in the hips and legs only, without backache. In a small proportion of cases the symptoms of motor weakness, numbness and tingling, and, rarely, paralysis of the legs may be referable to pressure on the cauda equina. As already stated, about 10 per cent of patients are unaware of the presence of this condition.

The backache, slight stiffness and weakness of the back, or pain in the legs is generally brought on by physical activity—hard labor, stooping and lifting—with relief by rest, especially recumbency. These symptoms are obviously not characteristic, indicating merely the existence of ligamentous strain, muscle spasm, stretching of, or pressure on nerve roots, such as may occur with a variety of low back lesions.

The objective examination may be much more revealing. In patients with gross displacements the diagnosis may be evident on inspection and palpation, because muscle spasm and the forward and downward displacement of the spine produce an exaggerated lordosis and a prominent spinous process and sacrum. Meyerding lists the findings which may be noted as follows: a shortened torso, prominent erector spinae muscles, broad appearing pelvis, list to the side, and the ribs may rest on or telescope into the pelvis. More frequently one finds merely the exaggerated lumbar curve on standing, absence of a posterior rounding of the lumbosacral curve on bending forward and on palpation, with the patient lying in the prone position a sharp dip forward from the posterior prominence of the upper sacrum into a hollow over the lumbosacral junction.

Röntgenographic study is indispensable to confirm the diagnosis and to establish the degree of deformity. Lateral views are, of course, most important in disclosing forward displacement. Meyerding describes the findings in the roentgenograms as follows:

Anteroposterior roentgenograms reveal the shortened lumbar spine, the superimposed fifth lumbar vertebra on the sacrum, the cocked up spinous processes, separation of the neural arch and spina bifida. In the lateral roentgenograms the degree of subluxation may be determined. One also notes the angle and width of the lumbosacral joint, the condition of the promontory of the sacrum (whether rounded or elongated) and the presence of slipping along the margins of the vertebral bodies. Sclerosing of the articular facets, the length of the neural arch, evidence of fracture or of congenital deformity such as separation, are all observations which influence one's decision as to the etiology and complicating factors present. A study of the roentgenograms can be carried out best in a moderate degree of light, and special attention should be paid to the contour of the spinal canal where the displacements are often more readily made out. The fifth lumbar vertebra is commonly found to be wedge shaped and the sacral portion elongated, concave and riding forward on the sacrum. The promontory

of the latter may impinge between the body and the posteriorly displaced spinous process.

Differential Diagnosis

In cases with gross deformity the clinical diagnosis, which is generally quite evident, requires only roentgenologic confirmation. In the milder grades of spondylolisthesis the symptoms may resemble practically every other type of lesion which may cause low back disability. Unless roentgenographic study is employed, with lateral roentgenograms included, the diagnosis may be readily missed. In some cases suspicion of a traumatic neurosis has been dispelled by roentgenographic evidence of spondylolisthesis. There remained no doubt then of the organic basis for persistent chronic low back disability. When manifestations of pressure on the cauda equina are particularly prominent, exclusion of the possibility of a protruded intervertebral disk or tumor of the cauda equina may be necessary first.

Treatment

A lumbosacral belt, reinforced by steel bars to support the lumbosacral spine, is all that is necessary for patients whose symptoms are mild. The belt is likewise satisfactory for patients who have no complaints, but need some supportive measure to offset the strains of their occupation. For women the lumbosacral brace may be incorporated in a high back front laced corset. Some patients, who follow laborious occupations and who are not relieved by supportive measures, may obtain relief by change of occupation, when feasible. Otherwise, surgical fusion of the spine must be resorted to.

Meyerding describes another aspect of treatment as follows:

In those cases in which spondylolisthesis occurs as a result of trauma and this is recognized immediately, an attempt should be made to reduce the deformity by traction and prevent its recurrence by casts. In cases in which the patient is placed in a recumbent position and traction, by means of Buck's extension, is applied, some improvement in the position of the vertebral bodies and relief of symptoms may be expected. With the legs elevated and at right angles to the thighs and with the thighs at right angles to the recumbent spine, the weight of the torso may be utilized in pulling the vertebral bodies into better position. The insertion of a Kirschner wire through the lower end of the femur maintains this position easily and the danger of irritation of skin is obviated. This position is maintained for six weeks, and following this a plaster cast may be applied, with the legs in extension, in the form of a double spica cast extending up to the axilla. This permits the patient to be moved about or turned over on his abdomen or side, by so doing the occurrence of pressure sores is avoided. The patient is kept in this cast for a further period of six weeks. At the end of this

time a lumbosacral support is applied and the patient is allowed up and permitted to walk with crutches

Surgical fusion of the spine must be resorted to in cases with severe disability and deformity. Young people (especially those whose occupations are strenuous and whose symptoms have persisted for years) unrelieved by conservative measures have no other recourse. A sufficiently large segment of the spine must be fused. The operative procedure required is a formidable one and has been described in detail by Meyerding (1938)

KISSING SPINES

Kissing spines a condition in which the spinous processes of the lower lumbar vertebrae are in apposition may be associated with low backache caused largely by ligamentous strain. Only very rarely do the spinous processes in contact form an actual joint which may be irritated or strained and cause pain. The condition is revealed by lateral roentgenograms of the spine. Relief may be obtained by restricting motion at the lumbar spine by a brace but when the cause of the pain can be attributed with certainty to impingement of the spinous processes resection or fusion of them is certain to afford permanent relief.

CONGENITAL ANOMALIES OF THE ARTICULAR FACETS

For a long time the gross and rather bizarre spinal abnormalities already mentioned usurped all of the clinical attention; this attention never even wandered to so near a structure as the intervertebral disk or articular facets. On *a priori* grounds alone it might have been surmised that since anomalies of the bodies and transverse processes are so frequently encountered analogous anomalies of the intervertebral articular facets might also exist. And indeed anatomical studies have quite frequently revealed various types of anomalous development of the articular processes. Such abnormalities are probably more significant in relation to low back and sciatic pain than some of the conditions so frequently suspected. Congenital variations in the shape and in the plane of the facets of the articular processes of the lower lumbar and lumbosacral joints may produce pain directly by pressure on nerve roots or by contributing indirectly to pathologic lesions and sprains at the intervertebral joints. This subject will be discussed in more detail further on (page 485).

It is time to time low back disabilities have been ascribed to congenital defects but the author agrees with Steindler who has said "in the last two decades certain morphologic factors principally of the type of anatomic

variation, have been variously clumed as producers of pain low in the back but in most instances no convincing causal connection could be established. In the final analysis, all congenital anomalies are now considered mainly as predispositional."

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CHAPTER XL

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

VI ABNORMALITIES AT THE SACRO-ILIAIC JOINTS

In the past abnormalities at the sacro-iliaic joints—arthritic or anatomic—have received more than their due share of attention. Many reasons have been advanced to support the claim that pathologic conditions at the sacro-iliaic joints are primarily responsible for low back and sciatic pain. Arthritic changes revealed roentgenographically and anatomically as lipping at the sacro-iliaic joints particularly at their inferior margins have constituted part of the evidence adduced. The possibility of separation, partial slipping and dislocation has also been invoked as a possible etiologic factor. Doubtless arthritic changes or mechanical defects resulting from violent trauma may occur at the sacro-iliaic joints as elsewhere. They may be responsible for some cases of low back pain—not many.

Sciatic pain may occasionally result from such lesions and also sciatic pain has been known to disappear following successful fusion of the sacro-iliaic joint. Despite these circumstances however, it is the consensus of opinion at present that sacro-iliaic abnormalities constitute a relatively infrequent cause of low back or sciatic pain.

In an analysis of the etiologic factors conducive to sciatic irritation Williams (1932) found only three cases or less than 4 per cent in which sacro-iliaic arthritis could be ascribed as the cause. In a subsequent study of 1000 cases of chronic or recurring low back pain with associated sciatic pain in the majority of them Williams (1937) stated that he found neither anatomic, roentgenographic nor clinical evidence to support the hypothesis that disorders of the sacro-iliaic synchondrosis were a primary etiologic factor.

It is difficult to reconcile the association of sciatic pain and sacro-iliaic disease on the basis of anatomic relationships between the sacro-iliaic joint and the sciatic nerve for contrary to certain prevailing impressions the sciatic nerve does not lie close enough to the sacro-iliaic joint to be involved directly by irritation from sacro-iliaic disease the nerve being amply separated from the joint surface by the piriformis muscle. Anatomic investiga-

tions concerning the relationship of the roots and trunks of the sciatic nerve led Danforth and Wilson to conclude that sciatic irritation is much less likely to occur at the sacro-iliac joint [than elsewhere] for there there is no canal nor semblance of a canal which holds the nerves against the joint. Any distension of the joint could cause apparently only a little lifting up of the nerve but could not in any way encircle it or exert pressure on it as the structures of the nerve are too easily displaced. Our personal conviction from examination of the specimens is that the chances for nerve involvement are much greater in the lumbosacral region than in the sacro-iliac region.

These views are to a large extent substantiated by Thompson's clinical observations on twenty three proved cases of sacro iliac tuberculosis. Upon analysis he found that characteristically the pain of sacro iliac disease was limited to the buttock or the posterior aspect of the hip region or radiated along the course of the sciatic nerve. Low back pain was absent except when active tuberculosis existed at the lumbosacral joint also. Only seven of his patients had sciatic pain—invariably on the side of the affected joint. Significantly of these seven five had in addition to the sacro iliac disease a large abscess in the buttock producing tension or inflammation in the surrounding structures. In two cases aspiration of the abscess brought about immediate disappearance of the sciatic pain. Two other patients complaining of sciatic pain did not have any infection in the buttock. Lumbosacral fusion for active tuberculosis cured one of these whereas sacro iliac fusion had previously failed. Although the other patient was completely relieved within the first few days after sacro iliac fusion it is doubtful whether the operation was responsible for this result fusion of the joint could not possibly have occurred in so short a time. On the basis of these observations Thompson logically concluded that sciatic pain is as a rule not produced by disease at the sacro-iliac joint.

For those who seek support for the diagnosis of sacro iliac subluxation through roentgenograms Prince related an experience at a meeting of eminent orthopedists at which he says "I was shown a roentgenographic demonstration of sacro iliac subluxation on the left side of the pelvis by a Bostonian and a sacro-iliac subluxation on the right side of the same pelvis in the same roentgenogram by a distinguished gentleman from Baltimore. This rather confirmed my growing doubts as to the certainty of our knowledge about sacro iliac lesions."

Today the pendulum has swung far away from the view that sacro iliac disease is the cause of most low back disabilities. I fear there is now some times an almost stubborn refusal to accept even obvious sacro iliac lesions as possible etiologic factors. Admitting that they are probably far less common than has been previously supposed we nevertheless do encounter cases

in which every indication points to the sacro iliac articulation is the sole cause of the low back complaint

When typical sciatic pain exists in association with sacro iliac disease it may be presumed that such pain results from concomitant muscular or ligamentous involvement about the sacro iliac gluteal or lumbosacral regions perhaps induced by secondary mechanical strain or muscle spasm Freiberg and Vinke and others have suggested that the sciatic pain is produced by spasm of the piriformis muscle induced by disease at the sacro iliac articulation Involvement of roots of the sciatic nerve may also result from postural deviations at the lumbosacral area these induced by the protective mechanism called into play to avoid pain at the sacro iliac joint

ETIOLOGIC FACTORS IN SACRO ILIAC DISEASE

Of course traumatic lesions at the sacro iliac joint—fractures or contusions—may occur Pitkin and Pheasant stressed the importance of sacro iliac slips in relation to painful affections of the upper sacral joints Their view is by no means universally or even widely accepted Violent trauma can of course produce definite sacro iliac luxation That is unusual however Fleming referring to the ethereal diagnostic flights as so-called sacro iliac subluxation said The anatomic position of the sacro iliac joint and the method of its protection are such that definite subluxation at this joint must be an occurrence of great rarity The unfortunate term subluxation has been inaptly applied to a condition of sacro iliac sprain The author is inclined to agree with this view

Infectious processes specific and nonspecific in nature may obviously settle in the sacro iliac joints as elsewhere We have seen infectious sacro iliac arthritis with disappearance of joint cartilage narrowing of the joint space and in some cases spontaneous fusion as part of a generalized atrophic arthritis Not infrequently a localized sacro iliac arthritis precedes the development of diffuse atrophic arthritis of the spine (Marie Strumpell's disease) In one such instance encountered surgical fusion of one sacro iliac joint was followed shortly after by extensive ankylosing spondylitis Such occurrences as these suggest that in a patient presenting the characteristic constitutional makeup and evidence of atrophic arthritis of the sacro iliac joints it is perhaps well to give some consideration to the possibility of impending ankylosing arthritis of the spine Such a patient should be especially watched in an effort to avert more widespread atrophic spondylitis Incidentally surgical arthrodesis of such sacro iliac joints may be wisely postponed in the expectation that spontaneous fusion will occur

CLINICAL MANIFESTATIONS

Typically, the patient with a purely sacro iliac lesion describes his ailment as "a pain in the hip." He points, however, to a region in the sacro iliac area corresponding to the upper border of the sacrospinous notch and describes a radiation to the buttock. There is frequently further extension of the pain to the posterior aspect of the thigh. Occasionally, however there is pain also low in the back, and, sometimes, typical sciatic radiation of pain, as in lumbosacral lesions. We have already alluded to possible explanations for the sciatic distribution of pain with sacro-iliac disease (page 427).

Localized tenderness over the inferior sacro iliac ligaments and along the upper border of the sacrospinous notch are important findings, particularly if they are unilateral, and if they correspond to the area of subjective pain.

Flexion of the spine (with the patient standing) is at first free then increasingly limited as the hamstrings become taut. The initial freedom of motion at the lower back is in contrast to the more complete fixation at the lumbosacral spine in conditions of comparable severity affecting the lumbosacral articulation. In the sitting position when the hamstrings are relaxed, forward bending may be quite free and unlimited in sacro iliac disease, yet impossible in active lumbosacral disease. Passive flexion of the lumbar spine, by flexing the hips and knees, is more free in sacro iliac than in lumbosacral disease. The Lasague (straight leg raising) sign may be positive only on the side affected.

Pain on hyperextension of the thigh, while the pelvis is fixed (Gaenslen's sign), is indicative of strain of the sacro iliac ligaments on that side. The patient lies supine on the examining table, so placed that one buttock projects over the edge of the table. The sacrum is fixed by flexing the other knee firmly against the abdomen, the patient holding it in place with his clasped hands. With one hand leaning on the patient's clasped hands, to insure complete fixation of the sacrum, the examiner depresses the leg, thus hyperextending the thigh which projects over the edge of the table. If there is involvement of the sacro iliac joint on that side, the hyperextension maneuver will produce pain. This procedure is then repeated on the opposite side.

Compression of the ilia and direct pressure over the lower end of the sacro iliac joint, through the rectum, may elicit pain when an active inflammatory process exists. Although all of these physical findings are most characteristic of sacro iliac disease, they may not be elicited. In the series of cases of sacro iliac tuberculosis reviewed by Thompson, the usually characteristic signs of sacro iliac disease were absent as often as they were

present. He found that straight leg raising and compression of the wings of the ilia for example elicited pain in only half of the cases.

DIAGNOSIS

The usual laboratory tests are generally of little aid in diagnosis. When destructive changes at the joint surfaces exist and tuberculosis is suspected the Mantoux (tuberculin) test may be of value. It is almost always positive when the arthritis is tuberculous; the possibility of tuberculosis may practically be ruled out if the test is negative. As is well known, however, a positive Mantoux test in an adult may be caused by antecedent tuberculous infection unrelated to the existing joint disease.

ROENTGENOGRAPHIC FINDINGS

Owing to the anatomic configuration of the sacro-iliac joint, pathologic changes in it are not always easily demonstrable in ordinary, flat roentgenograms. Stereoscopic views of the entire pelvis including the sacro-iliac joints are more informative. But even under such circumstances the roentgenograms may reveal nothing abnormal if there is a periarticular arthritis or if the symptoms are produced by purely ligamentous strain.

Destructive changes, particularly those localized to only a part of the joint as in tuberculosis or osteomyelitis are readily apparent. Equally evident are gross destructive effects of malignant neoplastic disease, either primary or secondary. In atrophic arthritis there may be evidence of diffuse atrophy and destruction of articular cartilage indicated roentgenographically by narrowing of the ribbon-like cartilage space. In advanced stages there may be additional evidence of sclerosis of bone surrounding areas of osteoporosis of hypertrophic changes at the joint margin, or of complete bony ankylosis. Though irregular lipping at the margin of the joint particularly at its inferior border is indicative of hypertrophic (osteo-) arthritis such arthritic changes may have no relation to the clinical symptoms, hypertrophic spurs at the margins of the sacro-iliac joints of elderly people exist not infrequently without producing pain.

With regard to the roentgenologic demonstration of sacro-iliac slips (subluxations) there is wide difference of opinion. Baetjer says "The so-called sacro-iliac subluxations do not exist. The joint is of the saw tooth variety and before a slipping could take place these saw tooth edges would have to be broken. These conditions must be ligamentous sprains."

The sacro-iliac articulation is one of the strongest in the body, and its anatomical structure is such that only the most severe trauma could cause it

to 'slip'." Darling agrees essentially with this point of view. But Chamberlain has described a roentgenographic technique whereby he feels he can demonstrate "sacro iliac slip" and sacro iliac relaxation. His diagnosis of such lesions is based chiefly on finding asymmetry of the pelvis associated with abnormal elevation of one pubis as compared with its mate.

TREATMENT

Although the specific cause of the condition may dictate special therapeutic measures, the general principles of treatment in sacro iliac disease are as follows:

Rest and immobilization are of primary importance. The more acute the process, the more urgent the need for rest. As we have stated, strains and sprains are the cause of a large proportion of acute painful disabilities at the sacro iliac joint, even when there is coexisting sacro iliac arthritis. The treatment of such sprains has already been described (page 449).

Rest in bed for a few days or a week or two, immediately after the onset of an acute episode of pain, is likely to be more effective than many weeks of rest later. A non sagging bed permits maintenance of the most favorable position, with the pelvis flat, and the normal lumbar curve supported by a small, firm pad placed in the hollow of the back. Additional comfort may be obtained by flexing the knees and supporting them by a pillow.

Fixation of the affected joint is provided by strapping with adhesive plaster, or by the application of a belt, or plaster shell. Although the strapping may have to be extended to include the lower lumbar spine, if there is concomitant involvement of that area, immobilization of the sacro iliac joint proper demands absolute fixation from below the level of the iliac crests to the coccyx. Immobilization may be effected by means of a plaster cast, this is cut and removed immediately after the plaster dries and then reapplied for definite periods daily.

Physiotherapy—chiefly, local heat applied with an ordinary baker—is of great value. For this reason the employment of casts and even strapping with adhesive plaster should be avoided when not absolutely essential. As the acute process subsides, massage is added. Later, exercises, especially those designed to strengthen the gluteus maximus muscle, are important.

Analgesics, chiefly salicylates, fortified with codeine when necessary, should be employed for additional relief of pain.

In cases of atrophic arthritis of the sacro iliac joint, focal infection should be removed when there is sufficient clinical indication to do so.

Manipulation is a therapeutic procedure practiced with great fervor by those who consider sacro iliac luxation (slipping) an important cause of sacro iliac sprains. In this country, Pitkin is one of the outstanding ex-

present. He found that straight leg raising and compression of the wings of the ilia, for example, elicited pain in only half of the cases.

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CHAPTER XLI

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

VII AFFECTIONS OF THE PYRIFORMIS MUSCLE

In 1928, Yeoman and, more recently, Freiberg and Vinke suggested that sciatic pain in association with sacro iliac disease may be induced through involvement of the pyriformis muscle

Freiberg and Vinke indicated that the pyriformis muscle actually bridges the sacro iliac joint and is in close anatomic relationship to it not only because of proximity, but also because a part of its origin is intimately bound up with the capsular investment of the joint and is, therefore, subject to reflex spasm consequent upon intra articular irritation, just as is observed in other joints "

With regard to certain anatomic and physiologic features bearing on the relationship of the pyriformis muscle to sciatic pain, these writers point out that,

The relation of the sciatic trunk to the pyniformis muscle is more or less unique It may pass either above or below the muscle, it may split and pass around the muscle or the muscle may be split and surround the nerve Again, there may be a splitting of both the muscle and the nerve, in which case any possible combination of the four parts may occur On fairly pliable cadavera the experiment was made of performing the straight leg raising test When the thigh reached approximately twenty five to fifty degrees of flexion with the trunk, the hand within the pelvis could plainly feel the tightening of the sacrotuberous ligament and of the pyniformis muscle as well It would seem that we have here the most plausible explanation of Lasegues sign or the straight leg raising phenomenon Many writers ascribe this to the stretching of the sciatic nerve In many patients with sciatic pain the limit of straight leg raising is reached when only a few degrees of hip flexion have been accomplished and long before stretching of the nerve may be spoken of

According to the theory of Freiberg and Vinke, sciatic pain may be expected in such cases because of the extremely close anatomical relationship between the pyniformis muscle and the sciatic nerve They argue, therefore, that any lesion which would produce spasm of the pyriformis muscle—

either a primary myofascitis or a reflex effect induced by lesions at the sacroiliac joint—could produce sciatic pain by mechanical pressure of the spastic muscle on the anatomically adjacent portions of the sciatic nerve. They add that direct pressure of the piriformis muscle on the nerve is not likely, that the effect may be produced indirectly through pressure of the spastic piriformis muscle on a large branch of the inferior gluteal artery and its accompanying vein which they found crosses the sciatic trunk under the belly of the piriformis. Continuous pressure here from contraction of the piriformis may conceivably produce a sustained congestion both in the vein and in the circulation of the nerve sheath. Thus might be explained not only the sciatic pain but also the tenderness in the piriformis area. Many facts adduced by Freiberg and Vinke substantiate the plausibility of their hypothesis.

More recently Freiberg has reported favorable results in the treatment of certain cases of sciatic pain by section of the piriformis muscle thus strengthening his theory concerning the role of the piriformis muscle in this syndrome. Even though further experience with myotomy of the piriformis muscle may substantiate a causal relationship between spasm of that muscle and sciatic pain the physician would still be under compulsion to search for those underlying factors in the lumbosacral and perhaps sacroiliac regions which might actually be the primary stimulus to spastic contraction of the piriformis. Freiberg realized this principle when he stated that whereas cases are observed in which the clinical study points to the piriformis muscle as the direct cause of pain, at the same time there is reason to believe that this is incidental to disease in the sacroiliac joint and that the sacrolumbar and even the lumbar segment of the spine may also be implicated. It would obviously constitute an inexcusable error to look on an operation on the muscle or fascia to relieve pain as fulfilling one's task and one's responsibility demands that this be followed by the mechanical and constitutional measures which are calculated to control the fundamental condition which has occasioned the pain.

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CHAPTER XLII

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

VIII CONTRACTURE OF THE ILIOTIBIAL BAND

In 1935, Ober presented data supporting the idea that certain cases of lame back, with or without sciatic pain—and with no roentgenographic evidence of change anywhere in the lower back region—may be caused by contracture of the iliotibial band

The iliotibial band is a portion of the fascia lata of the thigh and represents essentially a strong fascial extension of the tensor fasciae latae muscle. Thus, the iliotibial band extends from the crest of the ilium downward over the trochanter and outer aspect of the leg, being inserted into the outer tuberosity of the tibia.

Ober pointed out that in many patients with low back disturbances, 'the iliotibial band is extremely tight and prominent when the patient is lying on his back, with the knees together, or when he is in the erect position. The band is very rigid, almost bone-like in consistency, when under tension, usually about one half inch wide, and is raised above the level of the fascia lata, with which it connects anteriorly and posteriorly. It is easily located in a line, usually just in front of the trochanter. It may be situated immediately over the trochanter or it may be a little posterior. When the band is situated immediately over the trochanter, many patients complain of a snapping sensation in the hip. This snapping sensation is due to the riding of the band back and forth over the trochanter. When the contracture is present on one side, a lateral deviation of the spine is produced on that side and the pelvis is found to be tilted. The mechanics of the contracture and the leverage produced by the contracture are so great that it is possible that the unilateral contracture may account for irritation in the sacro iliac joint. When both bands are tight and in front of the trochanter, the lumbar spine is held in lordosis in both the standing and recumbent positions. If contracture is posterior to the trochanter, the spine is held in a lumbar kyphosis.'

The cause of such contracture of the fascia lata is not entirely clear. Ober pointed to certain postural attitudes during infancy as predisposing factors

In adult life the fascia lata probably becomes contracted as a result of many pathologic conditions particularly postural disturbances which are associated with excessive lumbar lordosis.

Examination in these cases Ober continued reveals limitation of straight leg raising and of forward bending at the lumbar spine. When these patients are asked to sit down and bend the body forward with the legs extended on the examining table it is very rare for the lumbar spine and pelvis to flex even to a right angle. Many of these patients are unable to stoop over and touch the floor with their hands.

Lili's sign may be elicited in most cases. While the patient is lying prone on the examining table the examiner flexes the leg on the thigh and as the flexion takes place the pelvis rises from the table.

According to Ober the most important sign of contracted fascia lata is the abduction test which he described as follows:

The patient lies on his side on a table, the shoulders and pelvis being perpendicular to the table. The leg on which he is lying is flexed at the knee and the hip is flexed and kept flexed to flatten the lumbar curve. If the patient is on his left side the examiner places his left hand over the patient's hip in the region of the trochanter to steady him. The right leg is flexed to a right angle at the knee and is grasped just below the knee with the examiner's right hand the leg and ankle being allowed to extend backward under his forearm and elbow. The right thigh is abducted widely and then hyperextended in the abducted position the lower part of the leg being kept level and care being taken to keep the hip joint in a neutral position as far as rotation is concerned. The examiner slides his right hand backward along the leg until it grasps the ankle lightly but with enough tension to keep the hip from flexing. The thigh is allowed to drop toward the table in this plane. (Caution: Do not bear down on the leg.) If the fascia lata and the iliotibial band are tight the leg will remain more or less permanently abducted. If the hip is allowed to flex or internally rotate the iliotibial band becomes relaxed and the leg falls from its own weight. The same procedure for the opposite side is followed in every case.

Ober stated that the abduction test is not always positive and that many persons who have no symptoms referable to their backs exhibit an abduction sign, Lili's sign and limitation of straight leg raising. He postulated that such individuals have the mechanical setup for the production of these symptoms which may be precipitated if there is an exciting cause.

When the contracture causes severe low back pain and sciatica Ober suggested treatment by fasciotomy. The method of procedure is as follows:

An incision is made from just below the crest of the ilium down to the tip of the trochanter directly over the contracted iliotibial band. The fascia lata is exposed forward as far as the anterior superior spine and backward to the edge of the gluteus maximus muscle. The area of the greatest contracture of the

fascia can be seen readily and felt easily. The fascia is now divided transversely from just below the anterior superior spine to the anterior border of the gluteus maximus muscle. There is immediate separation of the cut edges for a distance of from $\frac{3}{4}$ to $1\frac{1}{2}$ inches, depending on the amount of contracture present. If the operator now attempts to carry out the test described, it will be shown that the thigh will completely abduct.

In August 1937, Ober analyzed the results of fasciotomy in a total of 415 cases of low back pain and sciatica. This series included 75 cases operated on by Ober, and 340 cases operated on by other surgeons in various localities. Of the 415 patients, eighty-four (21 per cent) obtained no relief, seventeen (4 per cent) showed only partial relief, and 314 (75 per cent) had complete relief. The symptoms were relieved immediately or after intervals up to one year. The average time before relief took place was about three months."

Discussing the 84 cases in which there was no relief or recurrence of symptoms, Ober stated:

In most of the cases in which there was no relief there was either a new growth, bad arthritis or an anomaly of the spine. In cases in which there was a recurrence, the tight fascia on the opposite side was at fault or else an incomplete operation had been done, i. e., the intermuscular septums were not divided or the anterior portion of the fasciae was not, especially that around the tensor fasciae latae and the sartorius. In one of my cases it was necessary to go down to the rectus femoris before the fascia was freed.

According to my experience with chronic, longstanding disabilities referred to the lower part of the back it takes considerable time before pain and stiffness of the spine disappear after fascial division.

It is, of course, evident that in a certain proportion of carefully selected cases relief from distressing low back pain and sciatica has been secured by means of fasciotomy. On the other hand, the failures indicate the actual difficulty of selecting cases suitable for this operation. The diagnostic signs described by Ober have not always proved to be reliable. The abduction test is not invariably positive when there is contracture of the fascia lata. Since the abduction test may be positive when there are no symptoms of low back disability, a positive abduction test in the presence of such symptoms does not definitely indicate a relationship between the two.

Is contracture of the iliotibial band, when causing low back disability, always the fundamental pathologic disturbance? Probably not. Frequently, contracture of the fascia lata must be secondary to other disturbances—postural or otherwise—in the lower back, and the elimination of such etiologic factors by conservative, nonsurgical measures (chiefly physiologic rest) may bring about permanent cure when fasciotomy alone might be of only temporary benefit. One may also suspect that relief following fasciotomy on

the iliotibial band is at least occasionally the result of the rest imposed rather than of the operative procedure itself

The author has observed a number of patients who had been subjected to fasciotomy of the iliotibial band without relief of their symptoms some subsequently recovered through conservative treatment rest, and im mobilization My own feeling therefore is that before subjecting the pa tient to this surgical procedure we need indubitable evidence of the need for it and more accurate criteria than we now have for the selection of pa tients who will respond favorably to it Its indiscriminate employment is bound to lead to many inevitable failures which may detract from even the occasional successes possible The value of fasciotomy on the iliotibial band as a therapeutic measure for low back pain must not be appraised until sufficient follow up data is available in the cases operated upon in the past several years

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CHAPTER XLIII

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

IX ABNORMALITIES AT THE VERTEBRAL ARTICULAR FACETS

Anatomic specimens have frequently revealed congenital anomalies and arthritic changes in the vertebral articular facets. However, surprisingly little attention has been accorded these structures clinically as a possible cause of low back and sciatic pain, yet the articular facets constitute the only true joints in the vertebral column. When it is realized further that the posterior articulations between the fourth and fifth lumbar and the fifth lumbar and first sacral vertebrae form the posterior walls of the intervertebral canals for the fourth and fifth lumbar roots, the possible relationship between sciatic irritation and pathologic lesions at these articular facets is quite obvious. We must recall again that the canals mentioned are the smallest of the intervertebral canals, whereas the roots traversing them are the largest of the nerve roots, and that the fifth lumbar nerve root is directly anterior to the posterior articulation between the fifth lumbar vertebra and the sacrum (page 425).

In their general anatomic structure the intervertebral articulations are comparable to other joints. The surfaces of the articular facets are covered with cartilage and the articular capsules of these joints are lined with synovial membrane. Hence, arthritic changes here, whether from acute or chronic trauma, infection, or degenerative processes, lead to the same pathologic sequence of events as in analogous arthritic processes elsewhere. However, the disability resulting from involvement of the lower intervertebral joints is likely to be relatively severe because of involvement of anatomically related nerve roots. And since the lower lumbar and lumbosacral articulations bear the brunt of the strain of the entire spinal column, any existing pathologic process there must be greatly aggravated by motion of the spine and by constant pressure between adjacent surfaces of the facets, a pressure which would be increased still more by muscle spasm. It is not difficult to understand, then, how painful such a disability can be and why it may be so stubbornly resistant to treatment.

As early as 1911 Goldthwait considered the possibility of anomalous

placement of the articular facets in relation to low back pain, and Danforth and Wilson, as well as others, hinted at the possibility of such a relationship. Putti (1927) was the first, however, to lay special emphasis on this



FIG. 11—Acute traumatic destruction of the lumbosacral intervertebral disk. Note the constriction of the lumbosacral foramina (After Williams, *Journal of Bone and Joint Surgery*, 19 550, 1937)

etiologic factor in the syndrome of sciatic pain. He discussed in considerable detail the various pathologic processes at the articular facets which he had observed in relation to sciatic nerve root irritation. As for congenital anomalies at the facets, he pointed out that they may affect primarily the intervertebral foramen and secondarily the nerve root passing through it, either by altering its shape and reducing its capacity, or by localized arthritis induced by abnormal mechanics of the spinal column. He realized that inflammatory processes arising in the vertebral articulations may produce a similar effect. The swelling and deformity induced by such conditions modifies the shape and reduces the capacity of the foramen. Pain results from irritation and compression of the nerve within it.

The articular facets in anatomic relation to nerve roots of the lumbosacral plexus may also be affected directly or indirectly by other pathologic conditions in the lower spine

Williams emphasized the effect of *acute or chronic traumatic destruction of the lumbosacral intervertebral disk* on the articular facets and intervertebral foramina. The integrity of the intervertebral disk determines to a large extent the proper relationship between the bodies of the vertebrae and, therefore, also of the articular surfaces of the facets. Destruction of the disk permits the body of the vertebra above to settle and carry with it its inferior articular facets. The result may actually be a partial subluxation of the corresponding intervertebral joints. Such subluxation may cause constriction of the intervertebral foramina, pressure on nerve roots, and, consequently, sciatic pain (Fig. 117). The abnormal anatomic relationship between the facets thus established leads, furthermore, to postural strain and, eventually, to hypertrophic arthritic changes. If the nerve root has escaped damage from the constricted intervertebral foramen resulting from the subluxation, it may in time be affected by the osteoarthritic spurs or the inflammatory exudate, which encroach still further upon the lumen of the foramen. Such an injured articulation is, moreover, subject to superimposed effects of chronic infections and of acute and chronic sprains. These precipitate acute pain by adding (to the pre-existing mechanical derangement) the effect of cellular exudation or effusion of fluid into the intervertebral articulations.

CLINICAL RECOGNITION OF LESIONS OF ARTICULAR FACETS

The patient suffering from low backache or sciatic pain produced by a facet lesion may present a history of acute trauma with injury to the low back many years before. Or the backache may appear insidiously without evident cause, as in cases of chronic postural strain or arthritis affecting the intervertebral joints. There may be repeated attacks of pain in the lower back (diagnosed as *lumbago*) yielding to treatment with fixation. Later, pain may develop and radiate along the thigh and lateral aspect of the calf and ankle.

During acute attacks the symptoms are essentially those usually encountered in acute sprains of the back. Pain is present and is generally increased by spinal motions, jarring, coughing, or sneezing. The muscles of the back are in spasm and tender to pressure. Motion is limited by the spasm and pain. Sciatic scoliosis, a particularly common finding during acute attacks, may exist. There may be flattening of the lumbar spine or *kypnosis*, in which case, attempts at extension of the spine increase the pain. When the involvement is unilateral, extension and lateral flexion of the

spine toward the side affected increases the pain. The Lasegue sign is positive extension of the knee with the hip flexed may be limited and may induce pain at the affected intervertebral joint. When the spinal nerve root is involved such manipulation of the leg may also produce sharp pain along the course of the sciatic nerve. In acute stages hyperalgesia may sometimes be elicited over the lateral aspect of the calf or ankle. In chronic cases of long standing hyperesthesia or anesthesia may occasionally be found along the course of distribution of the root affected. The Achilles reflex may be diminished or absent. Muscle atrophy and mild degrees of muscle weakness may be encountered the latter rarely. In general then the clinical manifestations are quite similar to those associated with many other lesions at the lower back.

Roentgenologic evidence is necessary for certainty in diagnosis of lesions of the articular facets but abnormalities of the intervertebral joints and intervertebral foramina particularly of the lowest lumbar and lumbosacral articulations are not easily demonstrable in ordinary roentgenograms of the spine. Even stereoscopic roentgenograms may prove inadequate in determining changes that may have occurred. With this difficulty in mind Ghormley and Kirklin have described a roentgenographic technique yielding oblique views in which the various changes in the articular facets are more clearly and more accurately visualized. Not only are perfect roentgenograms essential but also experience in their interpretation. With such requisites satisfied Ghormley and Kirklin and Putti have found narrowing of the space between the articulating surfaces of the facets, marginal proliferation about their articulating surfaces (indicating hypertrophic changes and in many instances traumatic arthritis), fractures through the surfaces of the facets and increased or decreased radiability of the facets and their supporting structures. Such roentgenograms are of course also useful for study of the vertebral bodies, the intervertebral disks and the sacro-iliac joints.

In addition to constriction of the intervertebral foramen the roentgenogram may reveal hypertrophic arthritis with spurs projecting from the bodies of the vertebrae and narrowing of the intervertebral joint space. Although the arthritic changes at the bodies of the vertebrae may be so striking as to divert attention from the facet lesion the latter is more likely to be the actual cause of the symptoms.

Since the facets of the articular processes of the first four lumbar articulations are in a sagittal plane the articular spaces there may be pictured diagrammatically (as was done by Putti) as clear lines with definite borders and in almost perpendicular direction. This line does not appear in the articulation between the fifth lumbar and the sacrum because it is not normally directed on a frontal plane (Fig. 118).

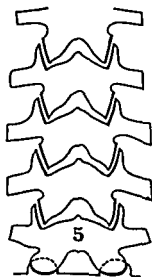


FIG 118

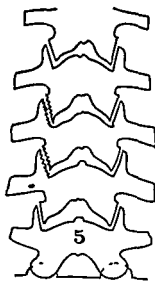


FIG 119

FIG 118 Radiographic picture of the articular system in the lumbar column under normal conditions. The articular facets of the first four vertebrae are placed on a sagittal plane while those between the fifth lumbar and the first sacral are in a frontal plane (After Putti *Lancet* 2 56 1927)

FIG 119 Schematic representation of the radiographic signs in lumbar arthritis (between second and third, third and fourth lumbar vertebrae) left side (After Putti *Lancet* 2 57 1927)

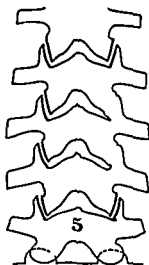


FIG 120

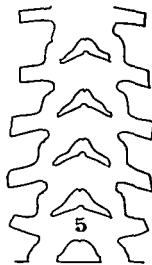


FIG 121

FIG 120 Partial ankylosis unilateral (between second and third, third and fourth lumbar vertebrae) (After Putti *Lancet* 2 57 1927)

FIG 121 Total ankylosis (After Putti *Lancet* 2 57 1927)

spine toward the side affected increases the pain. The Lasegue sign is positive extension of the knee with the hip flexed may be limited and may induce pain at the affected intervertebral joint. When the spinal nerve root is involved such manipulation of the leg may also produce sharp pain along the course of the sciatic nerve. In acute stages, hyperalgesia may sometimes be elicited over the lateral aspect of the calf or ankle. In chronic cases of long standing hyperesthesia or anesthesia may occasionally be found along the course of distribution of the root affected. The Achilles reflex may be diminished or absent. Muscle atrophy and mild degrees of muscle weakness may be encountered the latter rarely. In general, then, the clinical manifestations are quite similar to those associated with many other lesions at the lower back.

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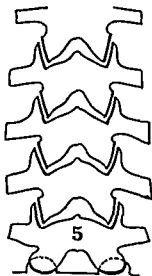


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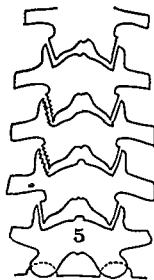


FIG 119

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FIG 119 Schematic representation of the radiographic signs in lumbar arthritis (between second and third, third and fourth lumbar vertebrae), left side (After Putti, *Lancet*, 2 57, 1927)

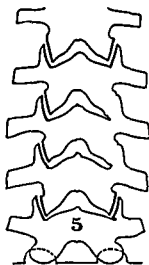


FIG 120

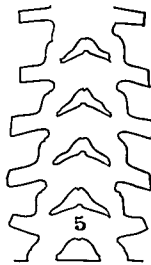


FIG 121

FIG 120 Partial ankylosis, unilateral (between second and third, third and fourth lumbar vertebrae) (After Putti, *Lancet*, 2 57, 1927)

FIG 121 Total ankylosis (After Putti, *Lancet*, 2 57, 1927)

spine toward the side affected increases the pain. The Lasegue sign is positive: extension of the knee with the hip flexed may be limited and may induce pain at the affected intervertebral joint. When the spinal nerve root is involved such manipulation of the leg may also produce sharp pain along the course of the sciatic nerve. In acute stages hyperalgesia may sometimes be elicited over the lateral aspect of the calf or ankle. In chronic cases of long standing hyperesthesia or anesthesia may occasionally be found along the course of distribution of the root affected. The Achilles reflex may be diminished or absent. Muscle atrophy and mild degrees of muscle weakness may be encountered the latter rarely. In general then the clinical manifestations are quite similar to those associated with many other lesions at the lower back.

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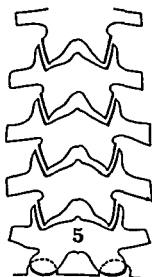


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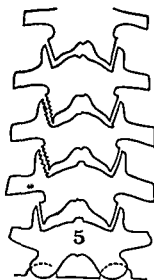


FIG 119

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FIG 119 Schematic representation of the radiographic signs in lumbar arthritis (between second and third third and fourth lumbar vertebrae) left side (After Putti *Lancet* 2 57 1927)

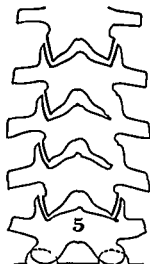


FIG 120

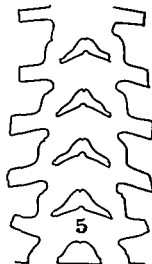


FIG 121

FIG 120 Partial ankylosis unilateral (between second and third third and fourth lumbar vertebrae) (After Putti *Lancet* 2 57 1927)

FIG 121 Total ankylosis (After Putti *Lancet* 2 57 1927)

spine toward the side affected increases the pain. The Lasegue sign is positive, extension of the knee with the hip flexed may be limited and may induce pain at the affected intervertebral joint. When the spinal nerve root is involved such manipulation of the leg may also produce sharp pain along the course of the sciatic nerve. In acute stages, hyperalgesia may sometimes be elicited over the lateral aspect of the calf or ankle. In chronic cases of long standing hyperesthesia or anesthesia may occasionally be found along the course of distribution of the root affected. The Achilles reflex may be diminished or absent. Muscle atrophy and mild degrees of muscle weakness may be encountered, the latter rarely. In general, then, the clinical manifestations are quite similar to those associated with many other lesions at the lower back.

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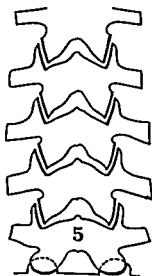


FIG 118

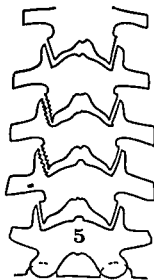


FIG 119

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FIG 119 Schematic representation of the radiographic signs in lumbar arthritis (between second and third third and fourth lumbar vertebrae) left side (After Putti Lancet 2 57 19-7)

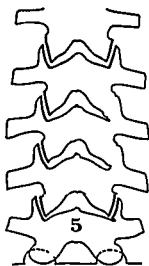


FIG 120

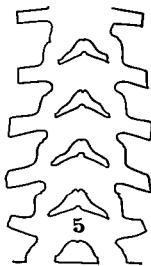


FIG 121

FIG 120 Partial ankylosis unilateral (between second and third third and fourth lumbar vertebrae) (After Putti Lancet 2 57 1927)

FIG 121 Total ankylosis (After Putti Lancet 2 57 19 7)

When the contour of the articular facets is distorted by arthritic pathologic changes, the roentgenograms will disclose the articular surfaces as lines not smooth and parallel, but irregular, jagged, and rough (Fig 119) The roentgenogram may also reveal localized ankylosis in one or another of the articulations (Fig 120) Or there may be total ankylosis with complete obliteration of the joint spaces (Fig 121)

It is obvious that such lesions in the articular facets cannot be discovered and will not be accorded the importance due them if consideration of them is entirely omitted, if roentgenographic study of the spine is limited to consideration of the bodies and transverse processes of the vertebrae, or if any but the most scrupulous care is given to the taking and interpretation of the appropriate roentgenograms

MEDICAL TREATMENT

Though in some cases operative measures may be required, in the majority of cases conservative therapeutic measures bring about amelioration or cure of the disability especially if applied in the earlier stages of the process

Rest (with or without fixation of the affected portion of the spine with plaster casts or removable supports) is the mainstay of the conservative regimen

When the condition is associated with marked muscle spasm and scoliosis and immobilization in a plaster spica is carried out, no attempt should be made to correct the spinal deformity before the application of the plaster, the scoliosis (nature's protective measure against pain) will resist correction until the muscle spasm has first been allowed to disappear through immobilization The spica should immobilize the pelvis as well as the trunk, otherwise it is useless It may be made removable so as to permit the application of heat and light massage The cast may be worn for two or three months, until muscle spasm has been resolved, after that a belt may be fitted to be worn for six months to a year

In cases in which the intervertebral foramen is narrowed as a result of destruction of the intervertebral disk, Williams recommended fixation with a plaster of Paris jacket applied with the lumbar spine flexed sufficiently to eradicate the lumbosacral lordosis In this way he aims to restore the width of the intervertebral space and to increase the diameter of the corresponding foramina When severe muscle spasm makes it impossible to obtain the desired flexion, resolution of muscle spasm may be obtained by the application of a cast for a short period After removal of this another is applied with the lumbar spine in the desired position of flexion

Rest may be required for weeks or months depending on the severity of the process. When the degree of disability is marked, and fixation with plaster is not employed, absolute rest in bed must be enjoined. The gains that have accrued during the periods of recumbency may be lost entirely by permitting the patient to break the periods of rest with several visits a day to the bathroom or one or two visits a week to the physician's office.

The eradication of focal sepsis and the improvement of the general physical state of the patient are important phases of treatment when the basic process is inflammatory in nature, but removal of focal infection alone does not constitute a cure. Questionable foci of infection should not be eradicated indiscriminately, the physician should have the clearest indication of the need for their removal.

Immobilization of the back is indispensable. Rest in bed and daily physical therapy may be supplemented by traction with Buck's extension if there is muscle spasm.

In some cases epidural injection of 40 to 60 cc. of a 1 per cent solution of procaine hydrochloride may be combined with immobilization.

In chronic cases, with spasm in the paraspinal and hamstring muscles, manipulation under anesthesia, followed by immobilization in a plaster spica cast may be advisable. Manipulation is, however, not devoid of danger in these cases. It is particularly essential to rule out protrusion of an intervertebral disk before manipulation is attempted lest more serious damage be inflicted.

When the patient becomes ambulatory he is to be fitted with a brace which maintains the spine in the corrected position. For those with postural abnormalities at the low back, the brace should be so designed as to maintain at the lumbosacral spine the normal lordosis attained by previous treatment. Such a brace should be worn for a period of six to twelve months though older patients may require much longer periods of support. For women, the brace may be incorporated in a front laced corset.

Physical therapy may be employed as a supplementary measure. Postural rehabilitation is one of the most important features of the treatment program in these cases, particularly in younger patients. It aims to establish normal physiologic realignment at the lower spine, and by maintaining such normal posture to eliminate the need for artificial supports and to prevent recurrence. Williams described in detail the exercises designed to correct the postural abnormalities generally encountered. These exercises aim to develop actively the anterior abdominal muscles, the gluteus maximus, and the hamstring groups, and to stretch the sacrospinalis and hip flexor muscles.

OPERATIVE TREATMENT

When adequate trial of conservative measures is ineffectual in bringing about relief consideration must be given to more radical, surgical measures. The latter are better adapted to and more clearly indicated in young patients who present a history of recurrent attacks of severe disability, not readily amenable to permanent cure by more conservative measures. But, as Ghormley emphasized, Failure of conservative treatment alone should not be an indication for operative treatment. He summarized the essential indications for surgical treatment as follows:

- 1 The patient must have persistent pain low in the back, with or without sciatic pain or recurrent attacks over a period of months.
- 2 The pain must be consistently localized over a definite area with tenderness on pressure over either the lumbosacral joint—that is, the space between the fifth lumbar spinous process and the first sacral spinous process, or laterally in the region of the lumbosacral articular facets—or he must have tenderness over one or both sacro-iliac joints. This tenderness is localized along the upper border of the sacrosciatic notch which is directly over the sacro-iliac joint. Such tenderness may be found also in the presence of lumbosacral lesions, in which event pain along the superior gluteal nerve is common.
- 3 Narrowing of the disk between the fifth lumbar vertebra and the sacrum must be demonstrated in the lateral roentgenogram.
- 4 Obliterative or destructive changes in the intervertebral articulations must be demonstrated by the oblique roentgenograms of the lumbosacral region.
- 5 The central nervous system should have been examined and found negative except for such evidence of irritation of nerve roots or of pressure on them as can be noted in many of these cases.

Technique

The surgical treatment aims to provide bony fusion of the involved segments of the spine and, by resection of a portion of the articular facet, decompression of the bony canal through which the affected nerve root passes.

For details of the operative technique, the original papers by Ghormley, Williams, and others should be consulted. Ghormley discussed the principles of the operative treatment as follows:

Production of lumbosacral ankylosis by bone graft or bony fusion cannot always be depended on to relieve these patients. In some cases even in which there was unquestionably excellent bony fusion or ankylosis sciatic pain persisted, this in spite of the fact that apparently the lumbosacral lesion was the original cause of the backache. Removal of the facet resulted in relief of the

sciatic pain. Such procedure alone can rarely be risked as a cure for sciatic pain. I have reported a case in which removal of a portion of the articular facet, thus enlarging the foramen of exit of the nerve, resulted in cure of severe sciatic pain.

The day may come when it will be possible to select all the patients whose trouble lies solely in the facet and who can be cured by its partial or complete removal. However, in the light of present knowledge, bony lumbosacral ankylosis must be produced, and, at the same time, sufficient bony and cartilaginous material resected from one facet or from both to remove a portion of the bony wall of the foramen through which the nerve root passes. Perhaps in many cases ankylosis of these joints will produce the desired result. I believe this is true in those cases in which backache only is the predominating symptom, but if sciatic pain is present in addition to the backache, in most instances enlargement of the foramen is essential, and this can be most easily accomplished by excision of the articular facet.

If operative treatment has been selected, the attempt should be made to determine exactly whether the pain is sacro iliac or lumbosacral. If the symptoms cannot be satisfactorily localized to one joint, two or even all three joints may be treated by operation. No sign is so consistently informative as elicitation of tenderness, consistently localized, on several examinations at varying intervals. Any case in which tenderness shifts is not a case for operative treatment.

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CHAPTER XLIV

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

X PROTRUSION OF LUMBAR INTERVERTEBRAL DISKS

In the past few years a number of studies have brought out the definite etiologic relationship between lesions of the intervertebral disks in the lower spine and low back and sciatic pain. Once the lesion is discovered and treatment is planned accordingly, progress is so marked and the results so gratifying that the physician cannot afford to ignore this relationship in any clinical consideration of sciatic pain.

PERTINENT ANATOMIC AND PHYSIOLOGIC CONSIDERATIONS

For clinical purposes, we may consider the intervertebral disks as composed essentially of a highly elastic fibrocartilaginous envelope, the *annulus fibrosus*, which contains a tense, gelatinous semifluid mass, the *nucleus pulposus*, situated approximately in the center of the disk.

The intervertebral disk furnishes support at the intervertebral portions of the spine, it keeps the vertebral bodies separated from adjacent segments and, by virtue of the elasticity of the *nucleus pulposus*, serves as an hydraulic shock absorber and equalizer. By maintaining the normal relationship between the bodies of adjacent vertebrae it also contributes indirectly to the maintenance of the normal anatomical relationship between the articular processes, controlling in this way the size of the intervertebral foramina (Fig. 112).

The lumbar intervertebral disks are anterior to the cauda equina and are separated from it only by the posterior longitudinal ligament and the meninges. Stretching across the posterolateral aspect of the spinal canal posterolaterally to the cauda equina on either side, are the *ligamenta flava*, connecting the laminae of adjacent vertebrae.

The lumbar subarachnoid space, a continuation of the subarachnoid space above, usually extends downward past the full length of the lumbar region, ending at the level of the lower border of the second sacral vertebra. The subarachnoid space is generally widest in its lumbar portion, narrowing

rather abruptly in its terminal sacral part. Occasionally, however, the subarachnoid sac ends much above its usual level. In this case the lower lumbar subarachnoid space may be much narrower than usual so that herniations of the fourth or fifth lumbar intervertebral disks occurring far laterally may fail to indent the cul-de-sac. Protrusions of the last lumbar intervertebral disks in such cases may not indent the shadow cast by lipiodol injected into the subarachnoid space.

Because of the elasticity of the nucleus pulposus the strains in normal movements of the spine can be counterbalanced with ease. Excessive strain on the intervertebral disk, however, during violent exercise such as jumping, bending or lifting may do real damage. A tear of the annulus fibrosus with extrusion of the nucleus pulposus may result or the nucleus pulposus may be driven into the body of the vertebra, a condition described by Schmorl.

THE LITERATURE ON THE SUBJECT

The necessary space for an exhaustive report on the literature on the subject is lacking here. The references given below may prove illuminating, however.

The occurrence of rupture of the intervertebral disk was described by Kocher as far back as 1896 and by anatomists, orthopedists and neurosurgeons at various times since then. Fibrocartilaginous nodules arising from the intervertebral disk and producing pressure on the spinal cord had been removed from various levels of the spinal column by neurosurgeons. Their interpretation of the nature and source of the lesion was not, however, always correct. The importance of this lesion in relation to the clinical syndrome of low back and sciatic pain had not been considered seriously until very recently.

In 1911 Goldthwaite reported a case of flaccid paraplegia of the legs which followed manipulation to reduce a presumed sacro-iliac strain. He concluded that the most likely explanation was a posterior displacement of the lumbosacral intervertebral disk with pressure on the cauda equina. In 1919 Avers emphasized the relationship between a narrowed intervertebral disk at the lumbosacral junction and low back and sciatic pain. In 1935 Williams wrote of reduced lumbosacral joint space in relation to this condition, inferring that its etiology was an injury to the lumbosacral intervertebral disk or destruction of it.

In 1934 Mixter and Barr reported nineteen proved cases of rupture of the intervertebral disk with protrusion into the spinal canal. They indicated that when the lesion occurred in the lumbar region pressure on one or more roots of the cauda equina produced symptoms quite similar to those of low back strain. A year later Mixter and Avers reported fifteen additional

cases of herniation or rupture of the intervertebral disk. And in 1937 Barr reviewed his experience with forty such cases occurring in the low lumbar spine and causing pressure on the cauda equina leading to sciatic pain. In 65 per cent of his cases the lesion occurred in the disk between the fourth and fifth lumbar vertebrae; in only 30 per cent in the lumbosacral region. Love and Walsh summarized their experience with 100 cases of protruded intervertebral disks in which operation was performed. They found herniation of the fourth lumbar intervertebral disk in 34 per cent of all protrusions and of the fifth in 41 per cent. The most recent reports are those of Spurling and Bradford (1939) and Love (1939).

CONSIDERATIONS ON THE ETIOLOGY AND PATHOLOGY OF INTERVERTEBRAL DISK LESIONS

The chief etiologic factor producing rupture of the intervertebral disk is trauma. Barr elicited a history of trauma in 77 per cent of the cases he studied. In some of his patients the disability followed immediately after the trauma; in others there was a latent period before the onset of symptoms. In some instances the injury so negligible in itself as to be forgotten may have nevertheless produced a slight tear or weakening of the annulus fibrosus. And Barr correctly assumed that the ordinary stresses of weight bearing alone were then sufficient to produce eventually a slowly enlarging herniation or prolapse of disk tissue. In Love and Walsh's series of 100 cases 32 per cent of the patients attributed the onset of their symptoms to a specific injury. Twenty nine per cent however could not recall any injury in relation to the onset of symptoms, indicating that if trauma played any part in the production of the lesion it was not of such magnitude as to leave any lasting impression.

One possible source of injury to the annulus fibrosus is suggested by Pease. He has observed twelve instances of damage to intervertebral disks due presumably to injury caused by too deep introduction of the needle during lumbar puncture. When such an extraneous source of possible injury does not present itself it must be assumed that rupture of the annulus fibrosus may occur when there is excessive acute traumatic compression of the disk, or when there is a point of weakness in the structure of the annulus fibrosus either from previous degenerative changes in it or from inherent anatomical defects.

Williams described in detail the effects of acute and chronic traumatic destruction of the lumbosacral intervertebral disk. He believed that chronic postural strain with its attendant trauma is responsible for many cases in which chronic degenerative changes lead to marked destruction of the disk. Under such circumstances narrowing or loss of intervertebral joint space

may follow and secondary effects on the intervertebral joints and their articular facets may supervene. With settling of the last lumbar vertebra partial subluxation of the facet articulations may occur leading in turn



A



B

FIG 12 A Drawing from an x-ray picture showing loss of lumbosacral disk, arthritic reaction and diminution in diameter of foramina formed by fifth lumbar and first sacral segments as compared to those formed by fourth and fifth lumbar segments, following a loss of the lumbosacral joint space.

B Roentgenogram of a man aged fifty-eight. Sciatica began at age of thirty. Recurring attacks for many years. None for the past ten years. Note loss of joint space and natural fusion. (After Williams *The Journal of the American Medical Association* 99:16 - 1932.)

to traumatic hypertrophic arthritic changes at the facets. Either one or both of these pathological conditions may produce distortion or constriction of the intervertebral foramina and pain by pressure on nerve roots traversing them (Fig 117).

We have already referred to this phase of the subject when we discussed lesions of the articular facets in the pathogenesis of sciatic pain (page 485). Loss of normal elasticity of the intervertebral disk and narrowing of the intervertebral joint space produce abnormal traumatic stress. And eventually osteoarthritic changes may develop in the bodies of the vertebrae involved. These changes may become quite pronounced, revealing themselves roentgenographically as large irregular spurs. In some cases these may fuse and produce a desired fixation of the spine with spontaneous relief from the distressing low back pain if pressure on nerve roots does not co-exist (Fig 122). Hypertrophic arthritis localized to two vertebral segments in the lower lumbar or lumbosacral spine may frequently be caused by injury to the corresponding intervertebral disk and should direct our attention toward the possibility of establishing that diagnosis.

When the intervertebral disk has been injured and the annulus fibrosus

ruptured, the nucleus pulposus may be partially or entirely extruded, along with part of the dense, fibrous tissue of the annular portion. This mass usually becomes herniated into the neural canal generally to one side or the



FIG 123 Photograph of a portion of a spine removed at autopsy. Pedicles have been cut near the vertebral bodies, thus removing the neural arch and the spinal cord. A posterior prolapse, measuring one centimeter in diameter, is present (After Barr, *Journal of Bone and Joint Surgery*, 19 337, 1937.)

other, and less frequently toward the center, where the strong longitudinal ligament of the spine is attached. Barr described the appearance of the lesion at operation "as a small rounded eminence, usually varying in size from that of a pea to that of a hickory nut. The mass has an appearance similar to wet, rolled up blotting paper, usually grayish or pinkish in color" (Fig 123). He described the histologic appearance of the protruded

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disk is that of normal disk tissue, with variations in appearance, however, depending on which portion of the disk predominates and on the age of the subject. 'If it is the annular portion, dense fibrous tissue with an occasional cartilage cell predominates. If it is chiefly nucleus pulposus, there is a loose granular reticulum the spaces of which are filled with mucoid material and with occasional poorly differentiated groups of cells which may be remnants of the notochordal cells or of the fibrocartilaginous cells. In some instances there may be present bits of bone or a small part of the posterior longitudinal ligament.' Varying degrees of edema occur, at times the swelling of the nuclear and annular elements of the protruded portion of the disk is very marked.

CLINICAL MANIFESTATIONS

Although prolapse of the intervertebral disk may occur without producing any symptoms, the nodule projecting into the vertebral canal frequently produces pressure on roots of the cauda equina and sciatic pain.

Viewing the pathologic changes that may occur (herniation of the intervertebral disk or its nucleus pulposus, disintegration of the disk, narrowing of the joint space, constriction of the intervertebral foramina, and secondary hypertrophic arthritis at the margins of the bodies of the vertebrae and at the facets of their articular processes) it is not difficult to understand the basis for the pain which may occur both in the lower back and along the course of the sciatic nerve.

We have already discussed the manifestations resulting from lesions at the articular facets and constriction of the intervertebral foramina. As we have stated these symptoms follow acute or chronic destruction of the disk with narrowing of the intervertebral joint space but without significant herniation of the nucleus pulposus. We shall, therefore, here concern ourselves chiefly with the clinical manifestations which result from *herniation* of the disk and pressure on adjacent structures.

The pain in the lumbosacral region probably results from the local effects of the lesion. The posterior longitudinal ligament may be irritated by pressure of a protruded disk or secondary arthritic changes that may ensue.

The sciatic pain (usually described as a deep seated burning or shooting pain, often excruciating in intensity) is the result of pressure on nerve roots of the lumbosacral plexus. Coughing, sneezing and change in position generally aggravate the distress. Some patients are somewhat relieved by rest in bed, others are more comfortable when standing or sitting. Nocturnal pain is not infrequent.

Intermittence of symptoms and recurrent episodes of sciatic pain are characteristic. Between attacks the patient may feel quite well. To explain

these recurrent episodes of pain and remission from symptoms, one must assume that the protruded disk may return into the intervertebral space at times and be extruded again at other times. That this may be the case has actually been demonstrated in the cadaver, as well as roentgenographically and on the operating table. Chamberlain and others have noted 'with drawal' of disk material from the spinal canal during flexion, and protrusion of the disk during extension of the spine.

In some cases the pain may be caused by edema of the protruded portion of the intervertebral disk. When conservative measures of therapy are employed, as rest in bed or immobilization by other means, the pain may disappear as the edema subsides.

Unilateral sciatic pain is most frequently encountered, although bilateral involvement is not unusual. At operation the ruptured disk fragment has been found to press on only one root in over half of the cases. In none of these are objective sensory changes noted. It is rare though possible for a protruded lumbar intervertebral disk to cause only low back pain without sciatic radiation. Low backache practically always accompanies sciatic pain, however. In addition to pain, the patient may complain of muscle weakness, cramps in the calves of the legs, or numbness.

Complete paraplegia may result from pressure on the cauda equina. It may also result from manipulation under anesthesia performed without suspicion of the existence of the intervertebral disk lesion. One of Barr's patients related a history of complete paraplegia following manipulation planned to reduce a 'sacro iliac subluxation'. This accident was, of course, the result of pressure on the cauda equina from a large protrusion of an intervertebral disk. One of the author's patients (from whom a protruded fourth lumbar intervertebral disk was removed) had a similar experience. She stated that one year previously she had had a manipulation of the lower back for severe 'sciatica', subsequently there developed flaccid paralysis of one leg, anesthesia, and loss of control of the bladder and bowel. At operation there was evidence of injury to the cauda equina at the level of the disk protrusion, indicated by the existence of a localized adhesive arachnoiditis which involved several caudal roots.

Sphincteric disturbances are relatively unusual, but when they are associated with sciatic pain they should arouse serious suspicion of a protruded disk. Disturbance of bladder function, accompanying severe sciatic pain, was the first arresting fact in the history of one of our patients suffering from a protruded disk.

There is usually a list of the lumbar spine (sciatic scoliosis), either toward the affected side or away from it. As a result of muscle spasm, the lumbar spine may present a kyphos which cannot be reduced by active or passive motion. Movement at the lower back is generally markedly restricted and

aggravates the pain. Tenderness to pressure may be noted either over the site of the intervertebral disk lesion, over the sacro-iliac joint region, or at the gluteal fold. Sciatic tenderness occurs in over half the cases. The Lasegue (straight leg raising) test is practically always positive, more markedly so on the side of the greatest pain. Very exceptionally, the neurologic examination may, however, be entirely negative.

Sensory changes, abnormal reflexes, and muscle atrophy may be noted in a small proportion of cases. Diminution or loss of the Achilles reflex, on one side or bilaterally, is one of the most common of the objective neurologic findings. Love and Walsh found diminution or absence of the Achilles reflex in 57 per cent of their cases, sensory loss in only 31 per cent. When present, such neurologic findings are, of course, of the greatest importance indicating the necessity for most intensive study, including lipiodol examination.

Roentgenographic study, to be of value in such cases, must include an teroposterior and lateral views of the spine. Even under these circumstances, normal roentgenograms are noted in about one half of the cases in which a lesion of the intervertebral disk exists. When positive roentgenograms may reveal narrowing or disappearance of the intervertebral joint space, constriction of the intervertebral foramina, and localized hypertrophic arthritis (Fig. 117). However, such roentgenographic findings do not establish the existence of prolapsed disk. There is, in fact, no consistent and dependable relation between a narrowed disk space and a protruded disk. Roentgenographic evidence of a narrowed intervertebral joint space, in a patient suffering from sciatic pain, demands further investigation to establish the presence or absence of protrusion of the disk. Thorough neurologic study, with examination of the spinal fluid and perhaps of the spinal canal with lipiodol is then essential. If examination reveals involvement of a single nerve root and narrowing or distortion of the corresponding intervertebral foramen by arthritic spurs, conservative treatment for the facet lesion is indicated. Such a course is particularly logical if the spinal fluid is found *perfectly normal*. Should such treatment not bring relief, examination of the spinal canal with lipiodol is necessary.

Roentgenographic evidence of a normal spine does not, however, exclude the possibility of prolapse of an intervertebral disk. We now feel that investigation of the spinal fluid and lipiodol examination are particularly necessary in such cases if the etiology of the sciatic pain remains obscure, especially if there is a history of acute trauma in relation to previous acute attacks of *'lumbago and sciatica'* and the condition has not proved amenable to conservative treatment. The presence of reflex or sensory changes in the lower limb furnishes still more reason for such study.

CHANGES IN THE SPINAL FLUID

Spinal fluid examination may add evidence of pressure on, and irritation of, the cauda equina, but may be disconcertingly unrevealing when the physician is groping for corroborative diagnostic data. Such was Barr's experience in a series of forty proved cases of prolapsed nucleus pulposus. *Examination of the spinal fluid revealed evidence of a dynamic block above the level of the needle in only four cases (10 per cent).* In the other thirty six cases the dynamics were normal.

Love has found his 'reversed' Queckenstedt test of great value in the diagnosis of intervertebral disk protrusion, when the spinal fluid examination is otherwise normal. On occasion, this test has given the only clue to the cause of the sciatica. The technique for the performance of this test is described by Love as follows:

The reversed Queckenstedt test is performed as is epidural injection, which is employed frequently in the treatment of sciatic pain. A caudal needle is placed in the sacral hiatus and care is taken to be sure the needle has not entered an abnormally low dura mater or a vein. A lumbar puncture needle is then inserted into the lumbar subarachnoid space, a manometer is attached and 10 cc. fractions of 1 per cent solution of procaine hydrochloride are injected into the caudal epidural space through the caudal needle. Normally there should be a progressive rise in the manometric readings as the caudal sac is compressed by the extradural procaine. Four fractions of 10 cc. each or a total of 40 cc. of 1 per cent procaine are injected extradurally. If a tumor or a protruded disk of sufficient size to obstruct the caudal sac is present, no increase in the manometric reading will occur. A 'block' on reversed Queckenstedt test will have been established. Experience in performing this test is necessary lest one be misled into thinking a block is present when one does not exist. In cases of ordinary sciatic pain not caused by pressure on the caudal roots from a tumor or an extruded disk the sciatic pain usually is exaggerated and then relieved on injection of each fraction. The order of events is first, pain from irritation of the nerve roots and posterior root ganglia and then, relief as the fluid diffuses and its anesthetic properties become manifest. In cases of compression of the caudal roots by a tumor or disk the pain is unbearable and the epidural injection will have to be discontinued. The sign, in our experience, has been pathognomonic of a mass encroaching on the domain of one or more caudal roots.

Barr found the total protein in the spinal fluid above 45 mg. per 100 cc. in thirty five of his forty cases. In 1937 he stated that until the previous year, 'a total protein below 40 mg. per 100 cc. was considered indicative of no pathology in the cauda equina and, therefore, in such cases lipiodol examination was not done. At the present time, if the patient's symptoms

and clinical examination are consistent and he is unrelieved by conservative therapy we do not hesitate to do a lipiodol examination even when the total protein is within normal limits (20 to 40 mg per 100 cc) We know that a negative lumbar puncture does not rule out rupture of the intervertebral disc Love and Walsh found the total protein content of the cerebrospinal fluid to be 40 mg per 100 cc, or more, in 80 per cent of their cases of protrusion of a lumbar intervertebral disk But that means that in 20 per cent the protein content of the spinal fluid was normal that is, less than 40 mg per 100 cc In other words, an increase in the total protein of the spinal fluid above 40 mg per 100 cc points to the probability of protruded disk, but the finding of a normal protein value (less than 40 mg) does not exclude the possibility of such a lesion

LIPIODOL EXAMINATION

When fairly conclusive clinical evidence of a protruded disk exists and an increased total protein content in the spinal fluid has been found, fluoroscopic and roentgenographic examination of the spinal canal after the injection of iodized oil serves primarily to confirm the diagnosis beyond doubt and to localize the exact situation of the protrusion In certain other cases when clinical evidence points strongly to the possibility of intervertebral disk protrusion but the spinal fluid is normal the role of lipiodol examination is more than corroborative It serves then actually to establish the diagnosis Barr found lipiodol examination 90 per cent accurate in localization of the level of the lesion in the forty cases studied The experience of others has been equally satisfactory

AIR AND OXYGEN MYELOGRAPHY

In such cases Chamberlain and Young have used air and more recently oxygen in place of lipiodol as the contrast medium for myelography In their large experience with this procedure they have found it very satisfactory and actually claim it to be the equal of myelography with iodized oil Perfect technique yielding roentgenograms with good detail and adequate contrast is however absolutely essential The advantages of oxygen myelography are obvious There is less danger of arachnoiditis such as may occur after the injection of lipiodol consequently oxygen myelography may be employed when the clinical indications are so meager that the use of iodized oil might not be justified Chamberlain and Young have discovered lesions which would have been missed without contrast myelography among such cases

Because such scrupulous roentgenographic technique is required myelography with air or oxygen has not been given the extensive trial it deserves but it is doubtful whether it can displace lipiodol as the most effective contrast medium available today.

There is a real need for the development of some new contrast medium which will have all the advantages of lipiodol and none of its potentially irritating qualities. It is hoped that such a substance will soon be found.

TECHNIQUE OF ROENTGENOSCOPIC EXAMINATION WITH LIPIODOL

The technique of roentgenoscopic examination with lipiodol is described by Camp (1937) as follows:

The lumbar injection is preferred. After injection the patient is placed in a sitting position on the fluoroscopic table for about one minute in order to permit all of the oil to gravitate to the cul de sac. This being done the patient is placed in the prone position on a tilting fluoroscopic table with the foot end of the table depressed to prevent cephalad excursion of the oil until the actual fluoroscopic observation is started. The shoulders are supported by padded shoulder rests. If the oil is seen to be low down in the lumbar canal the foot of the tilting table is gradually elevated and the shadow of the oil as it moves cephalad is observed carefully.

If the symptoms indicate that the lesion involves only the lumbar roots it may not be necessary to observe the movement of the oil much above the conus. However we recommend that the oil be followed to the cervical region in all cases in an effort to pick up multiple associated or unsuspected lesions. Since protruding disks are situated in the anterior portion of the spinal canal they produce their maximal filling defect when the patient is lying prone. In this position the heavy oil will gravitate to the ventral aspect of the subarachnoid space and is in close contact with the protruding disk. If a persistent defect is observed films should be made as quickly as possible without disturbing the position of the patient. The modern spot film devices for fluoroscopy and radiography of the stomach are ideal for this work since they permit a rapid change from fluoroscopy to radiography. This is extremely important for if one wishes to record on films the appearance of this fluoroscopic image which to our minds is the most significant part of the examination it is necessary to have some means to radiograph the opaque oil on the move.

Whether a defect is observed or not fluoroscopic examination should include observations in the prone, oblique and lateral positions. Only by this means is it possible to determine accurately the anterior or anterolateral position of a mass that indents the column of radiopaque oil. After the excursion of the oil has been studied in the foregoing positions we routinely have the patient sit up again for one minute in order to collect the oil in the cul-de-sac and then repeat the examination with the patient supine. Many protruded disks will not

produce a recognizable deformity when the patient is supine, nevertheless we feel that this part of the examination is necessary in order to determine confidently whether or not a tumor of the spinal cord is present. The presence of a tumor of the spinal cord in addition to a prolapsed disk is not impossible.

Defects in the Column of Radiopaque Oil

Because the protruded fragment of the disk is extradural, it will push against the column of radiopaque oil in the subarachnoid space, on the central or ventrolateral aspect, and indent it or displace it posteriorly and sometimes laterally. Complete obstruction of the column of radiopaque oil has been observed in the case of large protrusions. The classic filling defect is a sharply-defined, rounded indentation in the shadow of radiopaque oil on one side of the midline opposite an intervertebral disk. The extent of this defect is influenced naturally by the size of the protrusion. It is generally better defined in a prone-oblique position and may or may not be evident in the lateral view. In the majority of cases the defect is unilateral, but bilateral defects are not uncommon. We have also observed several instances of multiple defects indicating multiple protrusions. Generally there is no obstruction to the flow of the radiopaque oil, but there were four instances of partial obstruction and three of complete obstruction. In two of the latter cases the complete obstruction prevented the visualization of a second lesion of the adjoining disk.

In addition to the outline of the defect produced by the mass of the protruded disk, significant changes in the shadows of the nerve roots may be present at the level of the lesion. These consist of edema of one or more nerve roots, which may be recognized by a broadening of the negative shadow of the nerve root if it is outlined and by displacement or deformity of the shadows of nerve roots within the subarachnoid space. If the oil has extended into the extradural portion of the nerve sheath, displacement or deformity of this root may occasionally be visible. In a few cases the shadow of the oil in the extradural portion of a nerve sheath was terminated sharply, at the level of the protruded disk, suggesting pressure at this point. In some instances in which several nerve sheaths were visible it was noticed that there was persistent non filling of the sheaths on the side of the lesion; this is probably of significance and suggests pressure or edema of the nerve. However, nerve sheaths fill so inconsistently that lack of filling on one side cannot be depended on as a reliable sign of protrusion of a disk.

Because of the gradual fusiform narrowing of the terminal portion of the subarachnoid space and the proportionately larger spinal canal at the fifth lumbar space protruded disks at the lumbosacral junction will not produce as marked a filling defect as they would at higher levels. Since it is desirable to fill the cul-de-sac as completely as possible in order to visualize the maximal effect of any protrusion it may be necessary to raise the patient to almost a standing position.

It is important to inject large enough quantities—45 to 50 cc—of lipiodol in order to fill the lower dural sac. Incomplete filling of the lower

sac may not reveal small lesions with only a partial block and a small filling defect. It should be emphasized that the prone oblique position is best suited for visualization of disk protrusions since the defect generally occurs



FIG 124 Anteroposterior spot film of the low lumbar spine after injection of lipiodol. Note the filling defect on the left side at the level of the disk between the fourth and fifth lumbar vertebrae. This defect remained constant on repeated examinations. The rupture was found at operation at exactly this location, but it was not quite as large as the x-ray seemed to indicate. (After Barr, *Journal of Bone and Joint Surgery*, 19:359, 1937.)

anterior to the cauda equina. A small protrusion which may be visible with the patient in that position may be missed if the observations are made with the patient supine. Lateral views are less reliable since the defect (which as we have already indicated usually occurs at one side) may be obliterated by the lipiodol shadow on the opposite unaffected side (Fig 124). When disk protrusion is associated with thickening of the ligamenta flava, the lateral view of the lipiodol column may reveal both an anterior and posterior indentation, the former produced by the disk protrusion, the latter by the thickened ligamenta flava. When thickened ligamenta flava alone are present, the anteroposterior view of the roentgenogram may reveal bilateral indentations, and the lateral view only a broad posterior indentation of the lipiodol shadow.

Lipiodol study is best limited to cases in which other clues pointing

to a disk lesion have been obtained. Under such circumstances a larger proportion of positive results may be expected, and the lipiodol may eventually be removed during operative exploration of the spine.

There is, moreover, the exceptional instance in which the clinical history and the neurologic findings are indicative of the probability of an intervertebral disk protrusion, but in which the spinal fluid protein content is not increased and the roentgenoscopic examination with lipiodol is negative. The author has seen one such case in which the diagnosis was confirmed at operation and the patient was cured of severely disabling sciatic pain.

A woman forty-eight years of age, complained of two recent attacks of severe sciatica on the left side accompanied by pain in the lower back. The first attack had appeared five months previously, developing abruptly while she was walking. *There was no evident precipitating trauma. The pain was excruciating and extended from the left sacro iliac region, down along the posterolateral aspect of the left leg to the heel and the toes.* She had to be assisted to her bed, where she remained for five weeks. The pain persisted day and night, even when she lay quietly, and the slightest motion in bed was agonizing. During the earlier phase of this attack there was disturbance of bladder function, the patient being unable to void without catheterization. The attack was accompanied by marked muscle spasm in the lower back, with scoliosis.

Within two months most of the pain disappeared, but a sensation of tightness along the back of the left thigh and leg persisted. The patient was able to leave for a vacation within the next month, however, feeling fairly comfortable without the use of any support for the back, such as had been prescribed for her elsewhere.

Five weeks previous to her appearance at my office (nearly five months after the first attack), while still away from home, she developed recurrence of severe pain in the left lower back (in the region of the left sacro iliac joint) with severe pain radiating along the course of the left sciatic nerve. This time, too, there was no evident precipitating cause, such as trauma. The character of the pain and its distribution duplicated closely that which had occurred several months before, except that in the second attack the pain was not so intense and the function of the bladder was not affected. There were no paraesthesiae with either attack. Pronounced muscle spasm, with scoliosis, accompanied the second attack, as the first.

The second attack of sciatica required three weeks in bed. At this time the pain had abated enough to permit the patient to be out of bed. A dull pain persisted, however, over the left lower back and along the posterolateral surface of the left thigh and leg. In addition to the dull pain there was a sensation of *tightness along the back of the left leg and distortion of the lower back by scoliosis.* For two weeks she had managed to be up and about with the aid of a lumbar brace. Besides, she was under treatment with short wave diathermy and postural exercises.

The patient presented a history of numerous previous attacks of pain in the lower back without sciatica which had occurred intermittently since she was sixteen or seventeen years of age. None of these was severe however and they were generally attributable to physical exertion such as playing golf. A history of the patient's jumping off a moving street car when she was ten or twelve years of age was elicited. But that episode was apparently not followed by pain or other evidence of injury. The patient's general health was otherwise good. She had previously suffered frequent attacks of sore throat. Two years before the examination she developed pains about the left knee for which tonsillectomy was performed. *The history was otherwise irrelevant to the condition presenting.*

Physical findings. The general physical examination revealed nothing of note. There was slight flattening of the lumbar curve and a list of the lumbar spine toward the right unaffected side. The lumbar paraspinal muscles were in a moderate degree of spasm. Forward flexion and hyperextension of the lower lumbar spine as well as rotation toward the right produced pain to the left of the lumbosacral region nowhere else. There was no muscle atrophy but tenderness was elicited over the trunk of the left sciatic nerve in the upper posterior part of the thigh. The Lasague sign was markedly positive on the left side negative on the right. The reflexes in the upper extremities and the patellar reflexes were normal. The Achilles reflexes on both sides were absent. Objective sensory changes could not be demonstrated.

The laboratory data were entirely normal. Roentgenograms of the lumbar spine in the anteroposterior, lateral and oblique positions were normal as was the roentgenographic appearance of both sacro iliac joints.

In view of the history of two recurrent attacks of severe sciatica, the disturbance of bladder function during the first attack and the absence of the Achilles reflexes, a diagnosis of probable protrusion of a low lumbar intervertebral disk, with pressure on the cauda equina, was made.

Spinal fluid examination and roentgenographic study of the spinal canal after the injection of 5 cc. of iodized oil (this phase of the study was carried out by Drs. Love and Camp) revealed disconcertingly normal findings. The total protein in the spinal fluid was 40 mg. per 100 cc. the upper limit of the normal range. The lipiodol did not reveal a filling defect anywhere along the spinal canal.

Despite these negative data from spinal fluid and lipiodol examination the probability of an intervertebral disk protrusion with nerve pressure could not be abandoned in view of the rather significant history and objective neurologic signs indicating pressure on roots of the cauda equina.

Exploratory laminectomy appeared justified but before proceeding with it it was decided to observe the effect of further conservative treatment. The patient was put at rest in bed whereupon her pain began to subside. When significant improvement had occurred the patient was allowed up and around to observe the effect of limited activity. Whereas she had been quite comfortable in bed disturbing pain appeared again about 10 o'clock each morning. After two weeks more of observation during which time the pain continued an exploratory left hemilaminectomy was performed by Dr. Love. On removing the left lamina of

but rather increases the probability of a protruded disk underlying a hypertrophied ligament. Resection of such a thickened ligament leaving the protruded disk behind is likely to leave the disability with it—uncorrected.

TREATMENT

The treatment of destructive lesions of the intervertebral disk with narrowed joint space, secondary changes in the articular facets and nerve root irritation at the intervertebral foramen has already been discussed (page 490).

Surgical removal of the protruded portion of the disk is the desirable treatment when that condition is found. Love and Walsh described the operative procedure as follows:

The operative procedure consists of either extradural or transdural removal of the protruded portion of the disk through a laminectomy wound. Removal of the spines and laminae of two vertebrae provides adequate exposure for the removal of a single protrusion. In cases of multiple protrusions between adjacent vertebrae three spines and laminae must be removed. In one case of multiple protrusions one of us (Love) did a double laminectomy because one protrusion was at the lumbosacral space and the other was low in the thoracic region.

In performing laminectomy the articulating facets should be preserved. Resection of the ligamenta flava which are usually thickened in cases of protruded disks affords adequate exposure for the extradural removal of the lateral protrusions. In case the lesion presents in the midline it is best to approach it transdurally and in this event a wide laminectomy is not necessary. The iodized oil is carefully removed at the time of laminectomy.

No fixation of the spine by bone graft, cast or even belt is necessary following the removal of a protruded disk. In our series of 100 cases we have not found it necessary or advisable to carry out fusion.

The patients are treated postoperatively the same as after simple laminectomy for tumor of the spinal cord. They are kept in bed twelve days and allowed to leave the hospital on the fourteenth day and to return to their homes three weeks after the operation has been performed. They are advised to refrain from heavy lifting and straining for a period of three months.

The results of surgical excision of offending protruded disks are extremely satisfactory. Patients are relieved of pain and the neurologic manifestations disappear when permanent damage has not occurred from too long continued pressure on nerve roots. Recurrence of protrusion of cartilaginous fragments from an intervertebral disk at the site of previous removal of one protruded section of that disk may occur. Fortunately this appears to be unusual.

With the foregoing considerations in view investigation of patients with sciatic pain will establish a number of cases as instances caused by intervertebral disk protrusion, probably a much larger number than we now suspect

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woven bundles of white fibrous and yellow elastic tissues, with the former predominating. Areas of calcification were found in all specimens, evidence of an inflammatory process was lacking, leucocytic infiltration was absent. It appears most probable that trauma is the etiologic factor, particularly since there is no pathologic evidence of an inflammatory reaction.

Although thickening may occur in any of the lumbar ligamenta flava, it is noted most frequently in the ligaments connecting the fourth and fifth lumbar vertebrae.

THE LITERATURE ON THE SUBJECT

In 1913 Elsberg described hypertrophy of the *ligamentum flavum*, causing compression of the cauda equina. In that case the hypertrophy followed direct injury to the fourth and fifth lumbar vertebrae. Nothing pertaining to this entity appeared in the literature until 1931, when Towne and Reichert, apparently unaware of Elsberg's observation, reported two cases. In 1933, Abbott reported one case, in 1937 Spurling, Mayfield, and Rogers reported seven cases, and in 1938, Brown reported seven additional cases. A number of other cases, not yet reported, in which thickened ligamenta flava were found to be the sole cause of low back and sciatic pain are known to the writer.

ETIOLOGY

Trauma appears to be the basic etiologic factor in the production of hypertrophy of ligamenta flava, although no specific proof of this fact exists. In most cases the history of trauma is definite. In others, in which such a history is not elicited, it appears likely that some unrecognized trauma is involved. The hypertrophy, therefore, probably represents scar tissue repair following rupture (incurred by minor or severe injury) of some of the elastic fibers of the ligamenta flava.

CLINICAL MANIFESTATIONS

The symptoms are essentially those of pain low in the back with radiation of pain into one or both legs. In this respect the symptoms are indistinguishable from those in many other conditions associated with low backache and sciatic pain. The symptoms may begin suddenly with trauma, as, for example, while lifting a heavy object in a stooped position. There may then be temporary freedom from pain for days or weeks, with subsequent acute or insidious recurrence of similar pain leading to protracted

incapacity The pain may begin insidiously from the outset without any antecedent episodes of acute pain Some patients are partially relieved of pain by rest in the recumbent position, most of them feel better sitting or standing Coughing, sneezing, or straining at stool generally augments the pain, as would be expected, in view of the involvement of nerve structures in the spinal canal

In addition, other symptoms may exist referable to involvement of various roots of the cauda equina Thus, sexual impotence, disturbances in the function of the bladder or bowel, 'numbness' in the extremities or perianal regions, subjective motor loss (weakness) in the legs, and the like, may occur

The clinical picture as a whole, being essentially that resulting from compression of the roots of the cauda equina, is indistinguishable from that produced by a protruded lumbar intervertebral disk or tumor of the cauda equina

The objective findings are essentially like those seen in cases of protruded lumbar intervertebral disks, including various types and grades of neurologic manifestation of pressure on the lumbosacral roots

The roentgenologic examination generally reveals entirely normal findings Incidental spinal abnormalities may coexist, however

DIAGNOSIS

It is extremely difficult, if not impossible, to establish an accurate diagnosis of thickened ligamenta flava on the basis of the clinical history and physical findings alone If the neurologic examination reveals even slight evidence of some intraspinal lesion, the indications for study of the spinal fluid are clear A change in the hydrodynamics of the spinal fluid is, of course, not to be expected, unless the needle is inserted below the level of the lesion In the latter event, there may or may not appear evidence of a partial or complete block, depending on the degree of obstruction present An increase in the total protein content of the spinal fluid is generally noted, however On the whole, the indications for study of the spinal fluid and the positive observations to be noted in cases of thickened ligamenta flava are identical with those pertaining to protruded intervertebral disks For this reason the reader is referred to the preceding chapter for details concerning this phase of the diagnostic procedure

Conclusive proof of the presence and location of the lesion is afforded by roentgenographic study of the spinal canal after the injection into the subarachnoid space of 45 to 50 cc of lipiodol The details of the roentgenoscopic procedure, the interpretation of the observations, and the differentiating features between thickened ligamenta flava and protruded in

tervertebral disks have also been discussed in detail in the preceding chapter to which the reader is referred

TREATMENT

The diagnosis established the treatment of this condition is surgical. It entails excision of the thickened ligaments (through a laminectomy) with removal of the involved laminae. The surgeon should always be alert to the possible presence of a herniated disk opposite the thickened ligament; such protrusion will occasionally be found when roentgenographic evidence of its presence might have been lacking. Spinal fusion is unnecessary if the suspected intraspinal lesion has been found and removed.

PROGNOSIS

Prompt relief of symptoms follows excision of the thickened ligaments. The pain disappears almost immediately after operation. The neurologic manifestations too gradually wane and disappear unless permanent damage to roots of the cauda equina has resulted from overlong duration of pressure.

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CHAPTER XLVI

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

XII TUMORS—"RHEUMATISM REQUIRING MORPHINE"

Although sciatic pain caused by a variety of benign pathologic processes may be severe and stubborn, the use of morphine is, fortunately, rarely necessary for the relief of pain in such cases. It should, in fact, be sparingly employed even during the most acute phases in any of the rheumatic conditions. It should never be prescribed for relief of pain in chronic arthritic states. The ultimate consequences, through habituation, may be disastrous in a condition in which pain is so apt to be protracted. Fortunately, the more harmless analgesics suffice if combined with proper placement of the patient in bed, physiotherapy, and sedation. The combination of codeine and salicylates may be necessary during periods of acute distress. Morphine may occasionally be required immediately following manipulation of joints or surgical operations. When the severity of the pain, under ordinary circumstances, forces the continued use of morphine for relief, serious consideration must be given to the probability that a tumor (either benign or, as is the case more often, malignant) is the underlying pathologic basis for the pain.

The author's experience has practically always substantiated Hench's axiom that 'rheumatism requiring morphine' is probably caused by a neoplasm. On repeated occasions this axiom has served to orient me when otherwise I might easily have missed the diagnosis. In one instance, a young man with severe sciatic pain requiring morphine for relief was proved to have a *hemangioma pressing on the cauda equina*. In another case, the severe low back and sciatic pain, relieved only by morphine, was established to be the result of *retropentoneal sarcoma in the pelvis*. Another patient, a woman in her sixties, with severe sciatic pain relieved by morphine, had recently been examined elsewhere, a diagnosis had not been established, roentgenograms of the spine and pelvis, taken shortly before the patient came under our observation, had been found normal. The history and findings were essentially those of such low back and sciatic pain as could be caused by *lumbosacral hypertrophic arthritis*, but the necessity,

for frequent recourse to morphine led to the suspicion that she suffered from carcinoma. Inquiry disclosed a history of slight substernal distress on eating and a sensation of food passing into the stomach with difficulty. With suspicion sharply focused on the search for carcinoma as a basis for the low back and sciatic pain, further roentgenographic study was carried out, revealing esophageal obstruction from a mediastinal tumor which was also infiltrating the parenchyma of the lung. At this time, only two weeks after the previously normal roentgenographic observations were made, roentgenograms of the pelvis revealed evidence of metastatic carcinoma in the ilia and lower spine. The subsequent course confirmed the diagnosis of widespread malignancy. We could cite a number of such examples, quite similar in their general pattern, but those already mentioned suffice to emphasize the fact that must be borne in mind in this connection. Ryneerson and Slocum have cited similar cases in which the necessity for the use of morphine led to the correct diagnosis of neoplasm when previously the patients were treated for "rheumatism."

One is apt to think of tumor (as a cause of low back and sciatic pain) as a rarity hardly worthy of consideration, which, indeed, it is not. Some of these patients are, therefore, treated for weeks or months by physiotherapy or manipulation, at a time when the diagnosis could easily be established by roentgenograms of the spine and pelvis. The establishment of an early diagnosis is certainly desirable, if it only serves to relieve these patients of the unnecessary burden of such futile therapeutic measures.

In some cases even a serious suspicion of tumor cannot be confirmed at the first examination, the objective findings and the roentgenogram may then be normal, as was the case in one of the patients cited above. If the clinical facts indicate the possibility of tumor, repetition of the examination, including repeated roentgenographic study, may eventually reveal the evidence confirming the diagnosis.

The symptoms may resemble in every respect the ordinary low back and sciatic syndrome. The pain may be confined to the lower back or the extremities, or there may be combined low back pain with sciatic radiation to the legs. There may be a history of injury or strain of the back which may be misleading. There may be singularly few symptoms, or none at all, to suggest the primary site of the neoplasm. The original source of the tumor may, in fact, never be discovered. However, when the severe low back disability relegates the symptoms of the primary lesion into the background, careful inquiry into the history may lead one to discovery of the primary tumor.

The situation of the tumor and its nature vary widely. The tumor may be intraspinal in situation, it may involve the lower spine or any of the bones of the pelvic girdle; it may arise from the muscular or soft tissues

about the pelvis, it may be a neurofibroma of a single lumbar or sacral nerve root, it may be a diffuse neurofibromatosis, affecting also the trunk of the sciatic nerve. Again, it may originate in one of the pelvic or retroperitoneal structures in the lumbosacral region. Multiple metastatic foci in various parts of the lower spine or pelvic girdle may be responsible. Carcinoma of the breast, thyroid, and prostate, as well as hypernephroma, are particularly to be suspected as possible sources of metastatic carcinoma causing low back or sciatic pain.

TREATMENT

The treatment depends, of course, on whether the tumor is benign or malignant, on its situation, and its accessibility to removal. Metastatic tumors present of course only a hopeless outlook. In such cases relief of pain is the chief aim of treatment and for this purpose recourse to morphine is frequently necessary. Roentgenotherapy sometimes affords temporary alleviation of pain. Section of spinal nerve roots and chordotomy are sometimes indicated.

Benign intraspinal tumors causing pressure on the cauda equina and sciatic pain are amenable to excision with complete relief from pain. Neurofibromata, which constitute a large proportion of the benign tumors at the cauda equina, lend themselves especially well to removal with slightest residual damage to the remaining nerve structures. Being benign, they are not subject to recurrence, thus assuring permanency of the clinical cure.

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CHAPTER XLVII

THE CAUSES AND TREATMENT OF LOW BACK AND SCIATIC PAIN

XIII MISCELLANEOUS

In a previous chapter we have referred to a number of conditions which must be considered in the differential diagnosis of low back or sciatic pain. Although not an integral part of the text, a reminder that these factors exist is pertinent here. Hence the following discussion which must necessarily be sketchy.

FRACTURES

There is a distinct possibility that fractures of the accessory processes or of other parts of the vertebrae will occur following traumatic injury to the spine. We would therefore urge that complete roentgenographic study of the spine, including oblique views, be carried out when such a condition is suspected (Mensor).

DORSOLUMBAR SPRAINS

Sprains in the dorsolumbar region of the spine may involve the trunks of the twelfth dorsal and first lumbar spinal nerves. As a result of involvement of the first lumbar nerve, pain may occur in the sacro iliac and lumbosacral regions, erroneously suggesting disease at those areas. Carnett and Bates, and Judovich and Bates have stressed the importance of recognizing this condition.

Tenderness at the point of emergence of the first lumbar nerve trunk is evident paravertebrally, below the last rib. In addition, pain and tenderness generally exist posteriorly in an area over the upper half of the gluteal region, extending to the crest of the ilium above, and to the level of the *intertrochanteric line* below. Anteriorly, the pain and tenderness involve a narrow strip parallel to, and just above and below Poupart's ligament, and over a small area at the uppermost part of the inner aspect of the thigh.

Treatment includes immobilization of the dorsolumbar region of the spine by strapping, the employment of heat, correction of postural defects which may predispose the patient to such sprains. Injection of the first lumbar nerve trunk with novocaine or alcohol may be tried in more resistant cases.

PRESSURE FROM LESIONS IN THE PELVIS AND RECTUM

Aside from tumors of the pelvis, already discussed as possible etiologic factors, other pelvic abnormalities, including lesions of the rectum, are comparatively rare factors in producing low back and sciatic pain. The significance of prostatitis and displacements of the uterus in causing such pain has been exaggerated. We have found such a relationship to be very uncommon, although we have not failed to look for such conditions. They are, moreover, not difficult to find if routine pelvic and rectal examinations are practiced.

CONSTITUTIONAL DISEASES

Numerous constitutional conditions affecting the integrity of the skeletal structures, or the nerves, may produce symptoms referable to the low back or sciatic pain. We have already cited the possibility of low back pain produced by localized syphilitic spondylitis. Syphilitic infection may also be related to sciatic pain by causing diffuse arachnoiditis, a localized gumma, or diffuse meningovascular changes with involvement of the posterior roots. This may lead to "lightning pains" bearing a remote resemblance to ordinary sciatica. Sciatic pain may occur in gout or diabetes. Low back pain may result from osteoporosis associated with hyperparathyroidism or true osteomalacia. Low back pain, again, was the chief complaint in a case of osteitis deformans (Paget's disease) we observed recently.

TOXIC FACTORS

The peripheral neuritis produced by absorption of alcohol, arsenic, lead, and other neurotropic toxins must be considered in relation to the etiology of sciatic pain. An interesting association of sciatic neuritis with liver disease was recently reported by Lachtman. In five cases the symptoms of sciatic neuritis preceded the onset of clinical evidence of liver damage and jaundice.

PRIMARY (IDIOPATHIC) SCIATIC NEURITIS AND HERPES ZOSTER

Although formerly a frequent diagnosis, primary sciatic neuritis is a real clinical rarity. The author has seen it but seldom. In strict definition this diagnosis should be confined to involvement of the sciatic nerve by direct trauma, exposure, or some such factor. It may appear after a fall or severe strain. It has been known to follow exposure to wet or cold. Infection may be superimposed upon these influences, as in other types of neuritis. It may occur also as a part of a generalized polyneuritis. The causative factor may be obscure. In addition to the sciatic pain there may be pain low in the lumbar or sacral region. The clinical signs of neuritis can be elicited. The reflexes are usually diminished or absent. The condition responds to conservative medical treatment with rest, heat, and large doses of Vitamin B₁.

Herpes zoster, apparently a spinal ganglionitis, may be distributed along the course of various branches of the sciatic nerve, and is generally associated with the characteristic herpetic lesions in the skin.

VASCULAR DISEASES

In thrombo-angitis obliterans (Buerger's disease), or arteriosclerosis, sciatic pain may develop, apparently related to circulatory insufficiency in the trunk of the sciatic nerve. Karnosh described two cases in which the sciatic pain syndrome was attributable to sudden ischemia of the sciatic trunk. Surgical exploration revealed the true nature of the lesion in one of these cases. Craig and Ghormley mentioned patients under their care who seemed to have a spastic condition affecting the inferior gluteal (sciatic) artery, which caused the sciatic pain and was associated with coldness of the extremity. Following intravenous injection of typhoid vaccine, the temperature of the two legs became equal and the pain disappeared."

FUNCTIONAL NERVOUS STATES

Functional nervous states, in psychoneurotic individuals or in those of constitutionally psychopathic makeup, may so lower the threshold for pain as to lead to backache or sciatic pain. Although precipitated by some external factor, the pain may linger long after the effect of the extraneous cause has been removed.

Traumatic neurosis is an example of such a functional manifestation with low back pain engrafted upon a previously sustained back injury. The subjective symptoms may persist long after every indication of the actual

injury has disappeared. Persons of emotionally unstable personality make especially good candidates for such neuroses. Add to this susceptibility the strain of domestic and economic difficulties, suggestion of friends with whom the patient discusses his disability, long drawn out and varied treatment for the back injury, varied opinions regarding diagnosis obtained from different physicians, pending litigation, and similar circumstances—combine such circumstances and you have a condition fixed and stubborn to manage. The physician owes it to his patients to protect them from as many of these aggravating influences as possible.

Such a patient presents a rather typical appearance. He is obviously worried, extremely concerned about his condition, and apprehensive about the examination. He relates the history with disproportionate emphasis. He is evidently sincere in his desire to get well. One gets the impression that the complaints are genuine, at least subjectively. The examination generally confirms the impression of nervous instability. There may be evidence of vasomotor abnormalities, excessive sweating and cold clammy skin. Deep pressure over the muscles causes pain, but relocalization of painful areas is inaccurate. Muscle spasm and limitation of motion are generally absent. Motions at the lower back are either painless and free, or cause excessive discomfort. The examination as a whole reveals many incongruities between the subjective complaints and the objective findings. Roentgenograms of the spine do not help in diagnosis and may be downright confusing.

These patients are not malingerers. Although the disability is functional in nature, it is no less real to the patient himself. Realization of this fact will permit the physician to approach treatment in such a manner as to win the patient's confidence. Carnett and Bates in their discussion on "railway spine" described many borderline organic states which have been falsely labeled traumatic neurosis. Thoroughness of examination, unbiased approach to diagnosis, and an understanding of the patient's problems pave the way for successful therapy. Once such a relationship of confidence is established, the physician relies on properly applied psychotherapy rather than on physical measures for a cure.

MALINGERING

The malingerer complaining of low back pain can usually be apprehended without much difficulty. It is an unfair assumption, however, that the diagnosis of malingering can be made on purely circumstantial evidence. The accuracy of such a diagnosis is dependent upon certain positive clinical manifestations elicited through the history and most careful physical examination. Only through such means, as well as through acquaintance with the clinical picture presented by such patients, may one avoid the

serious error of assuming a patient to be a malingerer when he is actually suffering from some organic low back disability

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INDEX

INDEX

- Abdominal manifestations of rheumatic fever 297
- Acetylcholine iontophoresis in chronic arthritis, 236
- Acetylsalicylic acid, 302
- Achlorhydria, 28, 31
in atrophic arthritis 28 31
- Acute rheumatic fever See Rheumatic fever
- Acute suppurative arthritis, 342
bibliography on 347
symptoms of, 342
treatment of, 342
- Adhesive arachnoiditis, 405
- Aeroplane splint 195
- Allergic arthritis 353
bibliography on, 357
classification of, 19
manifestations of, 354
treatment of, 354
- Allergy, bacterial in pathogenesis of atrophic arthritis, 57
- American Rheumatism Association 6, 20
activities of 6
- Aminopyrine 152, 303
in treatment of atrophic arthritis, 152
in treatment of rheumatic fever, 303
- Amputation of toes in arthritis 230
- Amylodosis, 152
- Angina pectoris 362
- Annulus fibrosus, 495
- Anomalies of articular facets congenital 468
- Appendicitis, chronic as focus of infection in arthritis, 78
- Arachnoiditis 511
- Arm, nerve involvement as cause of pain in 399
- Arsenic in treatment of arthritis 152
- Arteriosclerotic 'claudication' of spinal cord, 406
- Arthralgia, 371
bibliography on, 374
- Arthritic constitution, 26
derechet, 208
- Arthritis
allergic See Allergic arthritis
associated with lymphogranuloma venereum, 346
bibliography on, 349
classification of, 19
clinical manifestations of, 346
associated with ulcerative colitis, 79, 346
bibliography on, 349
classification of, 19
atrophic See Atrophic arthritis
of brucellosis, 345
bibliography on, 348
classification of, 19
chronic rheumatic See Atrophic arthritis
classification of, 17
bibliography on, 19
climacteric See also Hypertrophic arthritis
constitutional, 352
classification of, 19
deformans See Atrophic arthritis, Hypertrophic arthritis
degenerative See Hypertrophic arthritis
focal See Focal arthritis
gonococcal See Gonococcal arthritis
gouty See Gouty arthritis
of Haverhill fever, 345
bibliography on, 348
classification of, 19
manifestations of, 345
hemophilic See Hemophilic arthritis
hypertrophic See Hypertrophic arthritis
infectious See Atrophic arthritis
infectious, specific, 18
classification of 18
meningococcal See Meningococcal arthritis
of meningococcus (cerebrospinal) meningitis See Meningococcal arthritis
metabolic See Metabolic arthritis
mixed forms of, 371
classification of 18
neuropathic See Neuropathic joint disease

INDEX

- Abdominal manifestations of rheumatic fever, 297
- Acetylcholine iontophoresis in chronic arthritis, 236
- Acetylsalicylic acid, 302
- Achlorhydria, 28, 31
in atrophic arthritis, 28, 31
- Acute rheumatic fever See Rheumatic fever
- Acute suppurative arthritis, 342
bibliography on, 347
symptoms of, 342
treatment of, 342
- Adhesive arachnoiditis, 405
- Aeroplane splint, 195
- Allergic arthritis, 353
bibliography on, 357
classification of, 19
manifestations of, 354
treatment of, 354
- Allergy, bacterial, in pathogenesis of atrophic arthritis, 57
- American Rheumatism Association, 6, 20
activities of, 6
- Aminopyrine, 152, 303
in treatment of atrophic arthritis, 152
in treatment of rheumatic fever, 303
- Amputation of toes in arthritis, 230
- Amyodoxyl, 152
- Angina pectoris, 362
- Annulus fibrosus, 495
- Anomalies of articular facets, congenital, 468
- Appendicitis, chronic, as focus of infection in arthritis, 78
- Arachnoiditis, 511
- Arm, nerve involvement as cause of pain in, 399
- Arsenic in treatment of arthritis, 152
- Arteriosclerotic 'claudication' of spinal cord, 406
- Arthralgia, 371
bibliography on, 374
- Arthritic constitution, 26
derelict, 208
- Arthritis
allergic See Allergic arthritis
associated with lymphogranuloma venereum, 346
bibliography on, 349
classification of, 19
clinical manifestations of, 346
associated with ulcerative colitis, 79, 346
bibliography on, 349
classification of, 19
atrophic See Atrophic arthritis
of brucellosis, 345
bibliography on, 348
classification of, 19
chronic rheumatic See Atrophic arthritis
classification of, 17
bibliography on, 19
climacteric See also Hypertrophic arthritis
constitutional, 352
classification of, 19
deformans See Atrophic arthritis, Hypertrophic arthritis
degenerative See Hypertrophic arthritis
focal See Focal arthritis
gonococcal See Gonococcal arthritis
gouty See Gouty arthritis
of Haverhill fever, 345
bibliography on, 348
classification of, 19
manifestations of, 345
hemophilic See Hemophilic arthritis
hypertrophic See Hypertrophic arthritis
infectious See Atrophic arthritis
infectious, specific, 18
classification of, 18
meningococcal See Meningococcal arthritis
of meningococcus (cerebrospinal) meningitis See Meningococcal arthritis
metabolic See Metabolic arthritis
mixed forms of, 371
classification of, 18
neuropathic See Neuropathic joint disease

Arthritis—(Continued)

- physical therapy in See Physical therapy in arthritis
 - pneumococcal See Pneumococcal arthritis
 - proliferative See Atrophic arthritis
 - psoriatic See Psoriatic arthritis
 - rheumatoid See Atrophic arthritis
 - of scarlet fever, 343
 - bibliography on, 348
 - classification of, 19
 - incidence of, 343
 - manifestations of, 344
 - treatment of, 344
 - senescent See Hypertrophic arthritis
 - septic, 107
 - of serum sickness, 301, 353
 - bibliography on, 357
 - classification of, 19
 - specific, noninfectious, 351
 - suppurative See Suppurative arthritis
 - sylphilitic See Syphilitic arthritis
 - of temporomandibular joint, 370
 - bibliography on, 373
 - traumatic See Traumatic arthritis
 - tuberculous See Tuberculous arthritis
 - of typhoid fever, 346
 - bibliography on, 349
 - classification of, 19
- Arthrodesis*, 178
- of hip, 283
- Arthropathia psoriatica* See Psoriatic arthritis
- Arthroplasty*, 1--
- Smith Petersen, 1-8, 209, 283
 - vitalium cup, 178, 209, 283
- Arthrotomy* for hypertrophic arthritis, 282
- Articular facets, vertebral* See Vertebral articular facets
- Asthenic habitus* in atrophic arthritis, 27
- Atrophic arthritis*, 81, 294, 301, 331, 351
- abnormalities in large bowel in, 28, 31
 - acetylcholine iontophoresis in, 236
 - achlorhydria in, 28, 31
 - acute onset of, 82
 - age incidence in, 81
 - agglutination of hemolytic streptococci in, 96
 - albumin globulin ratio in, 50
 - allergy in pathogenesis of, 57
 - bibliography on, 62
 - antistreptolysin in, 39
 - anxiety, effect of, on, 30
 - anxiety states in, 28, 30
 - appetite, impairment of, in, 81, 83
 - arrested, 105

Atrophic arthritis—(Continued)

- arthrodesis in, 178
- arthroplasty in, 177
 - vitalium cup, 178, 209, 283
- asthenic habitus in, 27
- autonomic nervous system imbalance in, 27
- bacteremia in, 36, 71
- bacteria in etiology of, 35
- bacteriologic studies in, 35
 - bibliography on, 42
- basal metabolic rate in, 50
- basic considerations in, 20
- bibliography on, 32, 108
- blood count in, 90
 - bibliography on, 109
- "calcium disturbance" in, 51
- candidate, typical, for, 25
- capillary circulation in, 27
- capsuloplasty in, 176
- capsulotomy in, 176
- carbohydrate of diet in, 28
- carditis in, 85
- cervical spine in, 88
- characteristic appearance of patient with, 83
- characteristic deformities in, 88
- cholesterol content of plasma in, 51
- chronic mode of onset in, 82
- cinchophen in, 107
- circulatory flow in, 51
- classification of, 18
- climatic effect on, 29
- clinical manifestations of, 81
- cold damp climate, effect of, in, 29
- colon in, 27, 31
- compresses, hot, in, 234
- confusion of, with gout, 321
- constitution, arthritic,
 - stigmata of, 26
- constitutional background for, 22
- constitutional predisposition to, 22, 23, 24
- constitutional vulnerability to, 22, 23, 24
- contrast baths in, 234
- course of, 81, 104
- cure in, 105
- debilitating illness, effect of, in, 30
- definition of, 21
- deformities, characteristic, in, 88
- deformities in See also Prevention and correction of deformities in chronic arthritis
- cause of, 168
- cervical spine, 183

Atrophic arthritis—(Continued)

deformities in—(Continued)

- elbow, 196
- feet, 222
- fingers, 203
- hand, 203
- head, 183
- hip, 208
- illustrated guide to recognition, prevention and correction, 183
- knee, 210
 - traction for correction of, 215
- lumbar spine, 191
- prevention of, 168
- shoulder, 192
- spine,
 - cervical, 183
 - lumbar, 191
 - thoracic, 187
 - thoracic spine, 187
- delayed rate of sugar removal from the blood in, 51
- dental infection and, 66
- diagnosis of, 81, 90, 107
 - bibliography on, 111
- diathermy in, 235
- diet, carbohydrate allowance in, 28
- dietary indiscretions, influence of, on, 105
- differential diagnosis in, 107
- digestive symptoms in, 81, 84
- digestive tract in, 27, 31
- dilatation and redundancy of colon, 28, 31
- economic handicaps, effect of, on, 30
- edema, peripheral, in, 50
- electrocardiographic findings in, 85
- emotional stress, effect of, on, 31
- endocrine abnormalities in, 58
 - bibliography on, 62
- erythrocyte sedimentation test in, 92
 - technique of, 93
- etiology of, 22, 32, 35, 41, 113
- exercise in, 237, 238
- experimental reproduction of, 40
- exposure to cold and dampness as cause of, 81
- extraneous predisposing influences in, 29
- fatigue in, 83
- fecal impaction occurring in, 134
- fever in, 83
- filament nonfilament count in, 90
 - bibliography on, 109
- fingers, spindle shaped, in, 86, 87

Atrophic arthritis—(Continued)

- focal infection in, 39, 65
 - bibliography on, 71, 79
 - Billings' observations on, 67, 70
 - general considerations in, 65
 - interpretation of concept of, 67
 - Rosenow's studies on, 67
- foci of infection in, 74
 - appendicitis, 78
 - bibliography on, 79
 - bone marrow, 71
 - cervicitis, 77
 - cholecystitis, 78
 - dental, 75
 - gall bladder infection, 78
 - genito urinary tract as one of the, 77
 - intestinal tract, 78
 - nasal accessory sinuses as, 76
 - nasopharyngeal lymphoid tissue as, 74
 - prostatitis, 77
 - renal infection, 77
 - seminal vesiculitis, 77
 - sinuses as, 76
 - tonsils as, 74
- forms of manifestations of, 18, 81, 113, 121, 125, 127
- glossitis in, 59
- gout confused with, 99
- hands in, 86
- heart lesions in, 50
- heat in, 232
- heliotherapy in, 241
- heredity in, 24
- histamine iontophoresis in, 236
- hypercalcemia in, 108
- hypcholesterolemia in, 51
- hypoproteinemia in, 135
- icterus, effect of, on, 106
 - bibliography on, 111
- incidence of,
 - age, 81
 - sex, 81
- inductothermy in, 236
- infectious foci, effect of, on globulin production, 50
- invalidism in, 88, 89
- jaundice, effect of, on, 106
 - bibliography on, 111
- laboratory data in, 90
- liver damage in, 49
- liver function tests in, 49
- low frequency currents in, 239
- manifestations of, 81
- massage in, 236
- menstruation, effect of, on, 105

Atrophic arthritis—(Continued)

- metabolic abnormalities in, 58
 - bibliography on, 62
- microtrauma, effect of, on, 31, 32
- nephritis in, 51
- nervous shock, effect of, on, 30, 31
- neurogenic abnormalities in, 60
- nomenclature, 20
- nonprotein nitrogen concentration in
 - blood in, 51
- nutritional disturbances in, 59
 - bibliography on, 63
- occupational therapy in, 239
- osteotomy in, 177
- overwork, effect of, on, 30
- paraffin, application of, hot, in, 233
- parathyroid disturbance in, 51
- pathogenesis of, 56
 - bacterial allergy in, 57
 - bacterial toxins in, 58
 - bibliography on, 61
 - endocrine abnormalities in, 58
 - bibliography on, 62
 - experimental arthritis bearing on, 57
 - infection in, 56
 - metabolic abnormalities in, 58
 - bibliography on, 62
 - neurogenic abnormalities in, 60
 - nutritional disturbances in, 59
 - bibliography on, 63
 - vitamin deficiency in, 59
 - bibliography on, 63
- pathology of, 39, 45, 114
 - bibliography on, 53
- peripheral circulation in, 51
 - bibliography on, 55
- pernicious anemia accompanying, 105
- physical therapy in See Physical therapy in arthritis
- pigmentation of skin in, 85
- plasma albumin in, 50
- plasma globulin in, 50
- postural rehabilitation in, 239
- pre arthritic stage of*, 84
- precipitating etiologic factor in, 35
 - bacteriologic evidence on, 35
 - bibliography on, 41
 - clinical evidence bearing on, 40
 - serologic studies bearing on, 38
 - virus as, 37
- precipitins in, 38
- pregnancy, anchoring effect of, on, 106
 - bibliography on, 111

Atrophic arthritis—(Continued)

- prevention of, 155
- prodromal symptoms in, 84
- prognosis in, 156
- psoriasis and, 105
- psychogenic aspects of, 28
 - bibliography on, 33
- psychogenic factors in, 28
- quiescent, 105
- radio active substances, therapy with, 241
- renal function in, 51
- renal involvement in, 51
- roentgenographic manifestations in, 97
 - bibliography on, 111
- roentgenotherapy in, 242
- scleroderma accompanying, 52
- sedimentation rate of erythrocytes in, 50, 92, 149, 175
 - bibliography on, 110
 - technique of, 93
- serologic studies in, 38
 - bibliography on, 43
- serum protein concentration in, 50, 135
- sex incidence in, 81
- short wave diathermy in, 235
- shoulder involvement in, 88
- skin changes in, 85
- spindle shaped fingers in, 86, 87
- spleen involvement in, 49
- static abnormalities in, 31
- streptococci in, cataphoretic velocity of, 67
- streptococci in etiology of, 36
- subcutaneous nodules in, 49, 85, 294
 - bibliography on, 54
- sugar removal from blood in, 27
- sulphur metabolism in, 51
- symptoms of, 81
 - prodromal, 84
- synonyms for, 18
- synovectomy in, 176
- synovial fluid in, 81, 96
 - bibliography on, 110
- sypilis accompanying, 105
- systemic exposure to heat in, 234
- systemic manifestations of, 81
- temporomandibular joint involvement in, 88, 370
- tenotomy in, 178
- thyroid activity in, 50
- torticollis in, 82
- transfusions of blood in, 134, 173
- trauma in, 31, 32

Atrophic arthritis—(Continued)

- treatment of, 129
 - aminopyrine, 152
 - arsenic in, 152
 - bee venom in, 143
 - bibliography on, 161
 - bibliography on, 157
 - blood transfusions in, 134, 173
 - bowel management in, 134
 - carbohydrate allowance in, 132
 - chaulmoogra oil in, 143
 - bibliography on, 162
 - chrysotherapy in, 144
 - agranulocytosis from, 146
 - albuminuria from, 146
 - aplastic anemia from, 146
 - bibliography on, 162
 - dosage, 148
 - hepatitis from, 145
 - mode of action of, 144
 - purpura from, 146
 - results of, 149
 - toxic reactions from, 144
 - prevention of, 147
 - treatment of, 147
 - in treatment of atrophic arthritis of spine, 150
 - in treatment of Still's disease, 150
- climatotherapy in, 153
- codeine in, 152
- colonic irrigations in, 134
- dechlorin sodium in, 153
- desiccated thyroid substance in, 152
- diet in, 132
 - bibliography on, 158
- drilling of epiphyses in, 154
- drugs in, 151
 - aminopyrine, 152
 - arsenic, 152
 - bibliography on, 163
 - codeine, 152
 - hydrochloric acid, dilute, 152
 - iodides, 152
 - iron, 152
 - morphine, 151
 - neocinchophen, 152
 - sabcyates, 151
- endocrine preparations in, 152
- estrogens in, 153
- fever therapy in, 141
 - bibliography on, 161
- foreign protein in, 143
 - bibliography on, 161
- general considerations in, 129

Atrophic arthritis—(Continued)

- treatment of—(Continued)
 - gold salts in, 144
 - agranulocytosis from, 146
 - albuminuria from, 146
 - aplastic anemia from, 146
 - bibliography on, 162
 - dosage, 148
 - hepatitis from, 145
 - mode of action of, 144
 - purpura from, 146
 - results of treatment with, 149
 - toxic reactions from, 144
 - prevention of, 147
 - treatment of, 147
 - in treatment of atrophic arthritis of spine, 150
 - in treatment of Still's disease, 150
- hydrochloric acid, dilute, 152
- iodides in, 152
- iron preparations in, 152
- morphine in, 151
- myochrysine in, 149
- neocinchophen in, 152
 - "new" remedies in, 153
- nutritional, 132
 - bibliography on, 158
- operative, 154
 - bibliography on, 163
- psychotherapy, 154
- removal of foci of infection in, 135
- rest in, 131
- sabcyates in, 151
- specific measures in, 137
- sulfanilamide in, 142
 - bibliography on, 161
- sulphur, colloidal, in, 138
 - bibliography on, 159
- sympathectomy in, 154
 - bibliography on, 163
- systemic, 131
- transfusions of blood in, 134, 173
- vaccines in, 139
 - bibliography on, 160
- vitamins in, 133
 - bibliography on, 158
- unhygienic surroundings, effect of, on, 30
- uric acid metabolism in, 51
- urticaria, ameliorating influence of, on, 106
- valvular disease in, 85
- variable course of, 23, 104
- variation in course of, 23, 104
- various foci of infection in, 74
- vasospasm in, 52

Atrophic arthritis—(Continued)

- virus in etiology of, 37
 - bibliography on, 43
 - vitalium mold arthroplasty in, 178, 209, 283
 - vitamin deficiency in, 27, 28, 31, 59
 - bibliography on, 63
 - weather, effect of, on, 105
 - bibliography on, 111
 - weight loss in, 83, 85
 - worry, effect of, on, 30
 - wrist involvement in, 86
 - x ray therapy in, 242
- Atrophic arthritis of the spine, 22, 113, 187, 472
- bibliography on, 120
 - chrysotherapy in, 150
 - clinical manifestations of, 114
 - diagnosis of, 117
 - etiology of, 113
 - gold salts in treatment of, 150
 - pathology of, 114
 - roentgenographic manifestations in, 117
 - treatment of, 118
- Atrophic spondylitis See Atrophic arthritis of the spine
- Auro thiomalate, sodium See Sodium auro thiomalate
- Autonomic nervous system imbalance in atrophic arthritis, 27
- Bacterial allergy in pathogenesis of atrophic arthritis, 57
- mutation, 66
- Baer maneuver, 440
- Baker for application of heat in chronic arthritis, 232, 233
- Banjo splints for fingers, 203, 207
- Bechterew's disease See Atrophic arthritis of the spine
- Bee venom in treatment of atrophic arthritis, 143
 - bibliography on, 161
- Bence Jones proteinuria, 420
- Bibliography, general, 527 See also under specific topics
- Bigelow and Lombard, survey of, 3
- Blood transfusions in treatment of atrophic arthritis, 134, 173
- Bowel management in treatment of atrophic arthritis, 134
- Brace, back, 190
- knee, 214, 220
- Brachial plexus, pressure on, 404
- British committee for the control of rheumatism, 6

Brucella abortus, 368

- Brucellosis, arthritis of See Arthritis of brucellosis
- Buerger's disease, 524
- Bursitis, 369
 - bibliography on, 373
 - calcaneal, 369
 - olecranon, 369, 370
 - prepatellar, 369
 - radiohumeral, 370
 - subacromial, 369, 378
 - subdeltoid, 369, 378
 - treatment of, 369
- Calcaneal bursitis, 369
- Capsuloplasty, 176
- Capsulotomy, 176, 221
- Cast, wedging of, 216
- Castex splints, 171, 198
- Cellulose compound splints, 171, 198
- Cervical collar, 186, 284
- Cervical rib and the scalenus anticus syndrome, 394, 404
 - anatomic considerations in, 394
 - bibliography on, 398
 - clinical manifestations of, 396
 - etiology of, 395
 - incidence of, 395
 - pathogenesis of, 395
 - treatment of, 397
- Cervical spine in atrophic arthritis, 88
- deformities of, arthritic, 183
 - hypertrophic arthritis of, 273, 403
 - pain in, nerve involvement as cause of, 399
- Cervical spine in hypertrophic arthritis, 273, 403
- treatment of, 283
- Cervicitis, as focus of infection in arthritis, 77
- Charcot joint, 355 See also Neuropathic joint disease
- Chaulmoogra oil in arthritis, 143
 - bibliography on, 162
- Chemotherapy in gonococcal arthritis, 332
- Cholecystitis as focus of infection in arthritis, 78
- Chronic arthritis See Hypertrophic arthritis or Atrophic arthritis
 - classification of, 17
 - hypertrophic pachymeningitis, 405
 - infective polyarthritis See Local arthritis
 - rheumatic disease See Rheumatic disease, chronic

- Chrysotherapy, 144
 agranulocytosis from, 146
 albuminuria from, 146
 aplastic anemia from, 146
 arthritis, in treatment of, 144
 bibliography on, 162
 dosage, 148
 hepatitis from, 145
 mode of action of, 144
 purpura from, 146
 results of treatment with, 149
 toxic reactions from, 144
 prevention of, 147
 treatment of, 147
 in treatment of atrophic arthritis of spine, 150
 in treatment of Still's disease, 150
 Cinchophen, 323
 in atrophic arthritis, 107
 Claw toe, 230
 Characteristic arthritis See Hypertrophic arthritis
 Climate, effect of, in atrophic arthritis, 29
 Climatotherapy, 153, 304
 Colchicine, 320, 321, 323
 Colloidal sulphur, 138
 bibliography on, 159
 Colonic irrigations, 134
 Committee for the control of rheumatism,
 American, 6
 British, 6
 Complement fixation test in gonococcal
 arthritis, 329, 330
 Compresses, hot, in chronic arthritis, 234
 Compression of crests of ilia, 419
 of spinal cord, 405
 Congenital anomalies of spine, 412, 463
 bibliography on, 469
 as cause of low back and sciatic pain,
 anomalies of articular facets, 468
 deep seated fifth lumbar vertebra, 464
 kissing spines, 468
 spondylolisthesis, 464
 Constitution, human, and atrophic arthri-
 tis, 22
 Constitutional arthritis See Hemophilic ar-
 thritis
 classification of, 19
 Constitutional factor,
 in atrophic arthritis, 22
 in hypertrophic arthritis, 252
 Contrast baths in chronic arthritis, 234
 Decholin sodium in treatment of atrophic
 arthritis, 153
 Deep seated fifth lumbar vertebra, 464
 Deformities in chronic arthritis See Pre-
 vention and correction of de-
 formities in chronic arthritis
 Degenerative arthritis See Hypertrophic
 arthritis
 Dental infection and atrophic arthritis, 66,
 75
 Diathermy in chronic arthritis, 235
 Diet in treatment of atrophic arthritis, 28,
 132
 bibliography on, 158
 Dorsal spine in hypertrophic arthritis, 276
 Dorsolumbar sprains, 522
 Drilling of epiphyses, 154, 283
 Drugs, in treatment of atrophic arthritis,
 151
 aminopyrine, 152
 arsenic, 152
 bibliography on, 163
 codeme, 152
 hydrochloric acid, dilute, 152
 iodides, 152
 iron, 152
 morphine, 151
 neocinchophen, 152
 salicylates, 151
 Dupuytren's contracture, 365
 bibliography on, 372
 treatment of, 366
 Elbow, deformities of, 196
 Encephalitis, 406
 Encephalomyelitis, 406
 Endocrine abnormalities in atrophic ar-
 thritis, 58
 bibliography on, 62
 Endocrine therapy in atrophic arthritis,
 152
 Epidural injection, 435, 491
 technique of, 437
 Equinus of feet, 222, 224
 Ergotamine tartrate, 368
 Erythema arthriticum epidemicum See
 Arthritis of Mowerhill fever, 345
 Estrogenic substance in treatment of
 atrophic arthritis, 153
 Exercise in chronic arthritis, 237
 Fallen arches, 86
 Feet, deformities in, 222
 causes of, 178
 prevention and correction of, 178
 treatment of, 179
 equinus of, 222, 224
 fallen arches, 86
 pronated, 222, 224

- Felt's syndrome, 49, 121
- Femoral nerve, 423
- Fever therapy in atrophic arthritis, 141
 bibliography on, 161
 in gonococcal arthritis, 332
- Fibrositis, 361, 389
 bibliography on, 371
 as cause of low back and sciatic pain, 460
 bibliography on, 461
 treatment of, 461
 etiology of, 362
 headache caused by, 362
 intercostal, 362
 intramuscular, 361
 manifestations of, 361
 pathology of, 363
 periarticular, 361
 stimulating pleurisy, 362
 tendinous, 361
 treatment of, 363
- Fingers, deformities of, 203
 spindle shaped, in atrophic arthritis, 86, 87
- Fiske's plaster shell technique, 433
- Focal arthritis 21 127
 bibliography on, 128
 clinical manifestations of, 127
 treatment of, 128
- Focal infection, 275, 303
 in atrophic arthritis See Atrophic arthritis, focal infection in
 eradication of, 438
- Foci of infection in atrophic arthritis See Atrophic arthritis, foci of infection in
- Foreign body arthritis 254
- Foreign protein therapy, 143, 439
 bibliography on, 161
- Formol gel reaction, 299
 technique, 300
- Fracture of spine, 522
- Fret skin reaction, 346
- Frozen shoulder, 383, 389
- Functional nervous states, 524
- Gaenslen maneuver, 440
- Gaenslen sign, 473
- Gaenslen test, 419
- Gall bladder infection in arthritis, 78
- Ganglion, 367
 bibliography on, 373
- Generalized myositis fibrosa, 363
- Glossitis in atrophic arthritis, 59
- Gold salts, 144 See also Chrysotherapy
- Gold sodium thiosulphate, 147
- Gonococcal arthritis, 107, 301, 326
 bibliography on, 334
 blood count in, 328
 classification of, 18
 clinical manifestations of, 327
 complement fixation test in, 329, 330
 conjunctivitis with, 328
 diagnosis of, 328, 330
 differential diagnosis in, 330
 gonococcus complement fixation test in, 329, 330
 incidence of, 326
 indocyclitis with, 328
 laboratory findings in, 328
 pathology of, 326
 roentgenographic findings in, 329
 sedimentation rate in, 328
 sore throat preceding onset of, 328
 symptoms of, 327
 synovial fluid in, 329
 tenosynovitis with, 327
 treatment of, 331
 chemotherapy in, 332
 fever therapy in, 332
 filtrates in, 331
 physiotherapy in, 331
 serums in, 331
 sulfanilamide in, 332, 333
 sulfapyridine in, 334
 vaccine in, 331
 upper respiratory infection preceding onset of, 328
- Gonococcus complement fixation test, 329, 330
- Gout, 99, 108, 301, 311, 331, 367
- Gouty arthritis, 311, 331
 arteriosclerosis with, 313
 bibliography on, 324
 blood count in, 319
 body weight changes in, 312
 case report, 313
 classification of, 18
 clinical manifestations of, 313
 colchicine in, 320, 321, 323
 definition, 311
 diagnosis of, 318
 differential diagnosis of, 321
 diuresis preceding acute attacks in, 312
 great toe involvement in, 317
 heredity in, 311
 high fat diets in, 320
 history, characteristic, in, 315
 hyperuricemia in, 311, 312, 314, 319
 incidence of, 313
 joint changes in, 316
 laboratory data in, 314, 319

Gouty arthritis—(Continued)

- metabolism in, 312
 - nephritis with, 313
 - renal colic accompanying, 318
 - renal function in, 312
 - roentgenographic findings in, 314, 317, 318
 - sedimentation rate of erythrocytes in, 320
 - simulating atrophic arthritis, 321
 - symptoms of, 315
 - therapeutic test in, 320
 - tophi in, 314, 315, 316, 318
 - treatment of, 321
 - cinchophen in, 323
 - colchicine in, 321, 323
 - compresses in, 321
 - diet in, 321, 322
 - hygienic measures in, 323
 - salicylates in, 323
 - vitamin B₁ in, 321
 - urate calculi in, 318
 - uric acid level in blood in, 311, 312, 314, 319
 - water and salt excretion in, 312
- Gouty diathesis, 311
- Gouty tophi, 314, 315, 316, 318
- Growing pains, 296
- Gynegrin, 368
- Hallux valgus, 226
- Hammer toe, 229
- Hand, deformities of, 203
- Haverhill fever, arthritis of. See Arthritis of Haverhill fever
- Head, deformities of, arthritic, 183
- Headache, 371, 403
 - caused by hypertrophic arthritis of cervical spine, 274
 - treatment of, 283
- Indurative, 362, 403
- Rheumatic, 275
 - treatment of, 283, 362, 403
- Heat, application of, in chronic arthritis, 232
 - baker for, 232, 233
- Heberden's nodes, 252, 264, 268, 279, 280
- Heliotherapy in chronic arthritis, 241
- Hemophilic arthritis, 352
 - bibliography on, 357
 - classification of, 19
 - treatment of, 353
- Hemorrhage into the spinal cord, 405
- Herpes zoster, 406, 524

- Hip involvement in hypertrophic arthritis, 272
 - treatment of, 283
- Hips
 - arthroplasty, vitallium mold for, 178, 209, 283
 - deformities of, 208
 - Smith Petersen arthroplasty for, 178, 209, 283
- Histaminase, 354
- Histamine iontophoresis in chronic arthritis, 236
- Horizontal sacrum, 464
- Human constitution and atrophic arthritis, 22
- Hydrarthrosis, idiopathic, 367
 - intermittent, 367
- Hydrochloric acid, dilute, in treatment of atrophic arthritis, 152
- Hyperparathyroidism, 108, 523
- Hypertrophic arthritis, 20, 21, 251, 264, 355, 402, 498
 - acetylcholine iontophoresis in, 236
 - arthrodosis in, 178
 - arthroplasty in, 177
 - vitallium mold, 178, 209, 283
 - bibliography on, 257, 262, 276
 - blood count in, 267
 - cartilage degeneration in, 259
 - cervical spine, involvement of, in, 273
 - headache with, 274
 - treatment of, 283
 - cholesterol content of blood in, 51
 - classification of, 18
 - clinical manifestations of, 264
 - compresses, hot, in, 234
 - contrast baths in, 234
 - course of, 266
 - deformity in, 266. See also Prevention and correction of deformities in chronic arthritis
 - cervical spine, 183
 - elbow, 196
 - feet, 222
 - fingers, 203
 - head, 183
 - hip, 208
 - illustrated guide to recognition, prevention, and correction, 183
 - knee, 210
 - traction for correction of, 215
 - lumbar spine, 191
 - shoulder, 192
 - spine, cervical, 183
 - spine, lumbar, 191

Hypertrophic arthritis—(Continued)

deformity in—(Continued)

spine, thoracic, 187

thoracic spine, 187

diathermy in, 235

dorsal spine involvement in, 276

etiology of, 251

constitutional factor in, 252

foreign bodies in, 254

infection in, 256

metabolic factors in, 255

neuropathic factor in, 255

obesity in, 253

trauma from faulty posture in, 253

trauma, occupational, 254

exercise, effect of, on, 265

exercise in, 237

filament nonfilament count in, 91, 267

focal infection in, 275

forms of manifestations of, 18

headache caused by, 274, 403

treatment of, 283

heat in, 232

Heberden's nodes in, 252, 264, 268,

279, 280

heliotherapy in, 241

hip involvement in, 272

treatment of, 283

histamine iontophoresis in, 236

inductothermy in, 236

joint mice in, 261

knees, involvement of, in 271

laboratory findings in 267

loose bodies in 261

loose cartilaginous bodies in 271

removal of, 282

loose joint bodies in, 271

removal of, 282

low frequency currents in, 239

lumbar spine involvement in, 276

malum coxae senilis, 272

treatment of, 283

massage in, 236

menopausal arthritis, 281

treatment of, 281

nomenclature, 251

occupational therapy in, 239

osteochondromata in, 261

osteotomy in, 177

paraffin, application of hot, in, 233

pathology of, 259

physical therapy in See Physical therapy
in arthritis

postural rehabilitation in, 239

Hypertrophic arthritis—(Continued)

prevention and treatment of deformities

in, 282

radio active substances, therapy with,

241

rheumatic headache in, 275

treatment of, 283

roentgenographic manifestations in, 267

roentgenotherapy in, 242

sedimentation rate in, 267

short wave diathermy in, 235

shoulder in, 273

of the spine, 273

treatment of, 283

stiff neck in, 273

treatment of, 284

symptoms of, 264

synovectomy in, 176

synovial fluid in, 261

synovial hyperplasia in, 261

synonyms for, 18

systemic exposure to heat in, 234

tenotomy in, 178

trauma in relation to, 264

treatment of, 279

bibliography on, 286

contrast baths, 280

correction of anemia in, 280

correction of deformities in, 282

drugs in, 280

eradication of focal infection in, 281

general measures in, 279, 280

mechanical supports in, 281, 286

paraffin applications in, 280

physiotherapy, 280

postural correction in, 281, 286

relief from trauma, 280

surgical measures in, 282

thyroid extract in, 280

weight reduction in, 281

villous synovitis in, 261

vitalami mold arthroplasty in, 178,

209, 283

x ray therapy in, 242

Hypertroemia, 311, 312, 314, 319

Hypoprotecemia in atrophic arthritis, 135

Icterus in atrophic arthritis, 106

Iliotibial band, contracture of, 481

bibliography on, 484

L.H.'s sign in, 482

Ober's sign in, 482

treatment of, 482

Ober fasciotomy in 482

Inductothermy in chronic arthritis, 236

Indurative headache, 403

- Infection, dental, in atrophic arthritis, 75
in etiology of hypertrophic arthritis, 256
sinus, in atrophic arthritis, 76
tonsillar, in atrophic arthritis, 74
- Infectious arthritis See Atrophic arthritis
specific, 107
classification of, 18
- Infectious spondylitis See Atrophic arthritis of the spine
- Inflammatory rheumatism See Rheumatic fever
- Injection of sciatic nerve, 438
- Intermittent hydrarthrosis, 367
bibliography on, 373
ergotamine tartrate in, 368
etiology of, 368
gynergin in treatment of, 368
synovectomy in, 369
treatment of, 368
- Intervertebral canals, 425
foramina, 425
- Intervertebral disk herniations, 405 See also Intervertebral disks, protrusion of
- Intervertebral disks, 487, 488, 497
acute traumatic destruction of, 487, 497
anatomic relations of, 427, 495
chronic traumatic destruction of, 487, 497
lumbosacral, traumatic destruction of, 487, 497
protrusion of, 439, 495
bibliography on, 513
case report of, 508
clinical manifestations of, 500
diagnosis of, 511
etiology of, 497
incidence of, 510
lipiodol examination for, 504
technique for, 505
literature on, 496
Love's reversed Queckenstedt test in, 503
myelography for, 504
technique for, 505
physiologic considerations in, 495
reversed Queckenstedt test in, 503
roentgenographic findings in, 502
spinal fluid examination in, 503
spinal fluid protein in, 503
symptoms of, 500
treatment of, 512
- Intestinal tract as focus of infection in arthritis, 78
- Introduction, 1
- Iodides in treatment of atrophic arthritis, 152
- Iron in treatment of atrophic arthritis, 152
- Irritation of diaphragm by pulmonary or abdominal disease causing shoulder pain, 406
- Jaundice in atrophic arthritis, 106
- Joint diseases, etiology of, 18
- Joint mice, 261, 271
removal of, 282
- Juvenile rheumatism See Rheumatic fever
- Kissing spines, 468
- Knees, deformities of, 210
pillows causing, 212
traction for correction of, 215
hydrops of, 25
in hypertrophic arthritis, 271
- Lasque sign, 418
- Leucemia, 420
- Ligamenta flava, 495
thickened, 439, 507, 511, 515
anatomic considerations on, 515
bibliography on, 518
clinical manifestations of, 516
diagnosis of, 517
etiology of, 516
literature on, 516
pathology of, 515
prognosis in, 518
symptoms of, 516
treatment of, 518
- Lipiodol examination of spine, 504
technique for, 505
- Loose joint bodies in hypertrophic arthritis, 271
removal of, 282
- Loose osteocartilaginous bodies, 261
- Love's reversed Queckenstedt test, 503
- Low back and sciatic pain, 411
anatomic relationships bearing on pathogenesis of, 423
bibliography on, 421, 431, 443, 526
causes of
anomalies of articular facets, 468
arthritis See Spine, arthritis of
articular facets, vertebral, abnormalities of, 485
Buerger's disease, 524
congenital anomalies, 463
constitutional diseases, 523
deep seated fifth lumbar vertebra, 464
dorsolumbar sprains, 522

Low back and sciatic pain—(Continued)

causes of—(Continued)

- facets, vertebral articular, abnormalities of, 485
- fibrositis See Fibrositis as cause of low back and sciatic pain
- fractures of spine, 522
- functional nervous states, 524
- herpes zoster, 524
- horizontal sacrum, 464
- hyperparathyroidism, 523
- iliotibial band, contracture of, 481
- intervertebral disk protrusion, 495
- kissing spines, 468
- ligamenta flava, thickened, 515
- malunion, 525
- muscular and ligamentous strains and sprains, 447
- neuritis sciatic 523, 524
- ostitis deformans, 523
- osteomalacia, 523
- osteoporosis 523
- overfatigue of muscles as 430
- Paget's disease 523
- pelvic lesions, 523
- peripheral neuritis, 523
- postural strain See Postural low back strain
- postural strain as, 430
- protruded intervertebral disks, 495
- psoas muscle, affections of, 479
- rectal lesions, 523
- sacralization of fifth lumbar vertebra, 464
- sacro iliac disease, 470
- sacro iliac joint abnormalities, 470
- spina bifida occulta, 463
- spondylolisthesis, 464
- strain of muscles as, 430
- thickened ligamenta flava, 515
- thrombo-angitis obliterans, 524
- traumatic neurosis, 524
- tumors, 519
- vascular diseases 524
- vertebral articular facets, abnormalities of, 485
- examination of patient with, 416
- general considerations on, 411
- laboratory investigation in cases of, 419
- Lasegue sign in, 418
- lumbosacral lesions causing, 417
- lumbosacral nerve roots in relation to, 424 425
- medical investigation in cases of, 416
- myelography in cases of, 420

Low back and sciatic pain—(Continued)

- neurologic study in cases of, 420
- pain, character of, 414
- pathogenesis of, 411, 423
- bibliography on, 431
- peripheral innervation in relation to pathogenesis of, 429
- physiologic relationships bearing on pathogenesis of, 423
- roentgenographic investigation in cases of, 420
- sacro iliac lesions causing, 417
- sciatic nerve roots in relation to, 423
- spinal fluid examination in cases of, 420
- strain of muscles as cause of, 430
- symptoms, general character of, 414
- therapeutic test in cases of, 421
- treatment of, 432, 449, 455, 458, 461, 467, 475, 480, 482, 490, 492, 512, 518, 521, 523, 524, 525
- bibliography on, 443
- epidural injection in, 435
- technique of, 437
- fixation in, 433
- Fiske's technique, 433
- focal infection, eradication of, 438
- foreign protein therapy in, 439
- manipulation in, 439
- mechanical appliances in, 432, 434
- operative, 441
- paravertebral injection in, 438
- physiotherapy in, 435
- postural exercises in, 435
- rest in, 432
- roentgenotherapy in, 439
- sciatic nerve, injection of, in, 438
- surgical, 441
- Low frequency currents in chronic arthritis, 239
- Lumbago 82, 363
- Lumbar nerve roots anatomic relations of, 425
- Lumbar spine, deformities in, 191
- in hypertrophic arthritis, 276
- Lumbosacral articulation, 423
- Lumbosacral lesions, 417
- Lumbosacral plexus, 424, 425
- Lymphogranuloma venereum, arthritis of See Arthritis associated with lymphogranuloma venereum
- Malignancy, confused with arthritis, 151
- Malunion, 525
- Malum coxae senilis, 272
- treatment of, 283

- Manipulation, 475, 501
 of back, 439
 of shoulder, 196
- Mantoux test, 339
- Marie Strumpell disease See Atrophic arthrititis of the spine
- Massage in chronic arthritis, 236
- Meningococcal arthritis, 343
 bibliography on, 348
 classification of, 19
 treatment of, 343
- Menopausal arthritis,
 bibliography on, 287
 treatment of, 281
- Metabolic abnormalities in atrophic arthritis, 58
 bibliography on, 62
- Metabolic arthritis, classification of, 18
- Metabolic factors in hypertrophic arthritis, 255
- Metastatic malignancy and arthritis, 151
- Metatarsal bar on shoe, 225, 228
- Metatarsal pad, 225, 228
- Methyl glucamide of auro thio diglycollic acid, 148
- Miscellaneous rheumatic conditions, 361
 arthralgia, 371
 arthritis of temporomandibular joint, 370
 bursitis, 369
 Dupuytren's contracture, 365
 fibrositis, 361
 ganglion, 367
 intermittent hydrarthrosis, 367
 mixed forms of arthritis, 371
 myositis, 366
 snapping or trigger finger, 367
 synovitis, 370
 tenosynovitis, 366
- Mixed forms of arthritis, 371
 classification of, 18
- Muscular and ligamentous strains and sprains, 447
 bibliography on, 451
 causing low back and sciatic pain, 447
 pathology of, 448
 symptoms of, 447
 treatment of, 449
- Muscular rheumatism, 361, 366
- Mutation, bacterial, 66
- Myalgia, 361
- Myelography, 420, 504
 technique for, 505
- Myeloma, 420
- Myochrysine, 149
- Myofascitis, 361 See also Fibrositis
- Myositis, 361, 366
 bibliography on, 372
 chronic, generalized, 363
 fibrosa, generalized, 363
- Neominchophen in treatment of atrophic arthritis, 152
- Nerve involvement as a cause of pain in the cervical spine, shoulder and arm, 399
 bibliography on, 406
- Nervous states, functional 524
- Neuralgia, 404
- Neuralgic pains, 32
- Neuritis, 394, 399, 404
 peripheral, 523
 sciatic, 524
- Neurofibroma, 404
- Neurogenic abnormalities in atrophic arthritis, 60
- Neuropathic arthritis See Neuropathic joint disease
- Neuropathic joint disease, 355
 bibliography on, 356, 357
 classification of, 19
 clinical manifestations of, 355
 treatment of, 356
- Neurosis, traumatic, 415, 524
- Neurosiphilis, 406
- Neurotrophic factors in etiology of hypertrophic arthritis 255
- Nodules, subcutaneous, 85, 294
- Noninfectious specific arthritis 351
- Nonspecific protein shock, 304
- Nucleus pulposus, 495
 extrusion of, 499
- Nutritional disturbances in atrophic arthritis, 59
 bibliography on 63
- Nutritional management in atrophic arthritis, 132
 bibliography on, 158
- Ober sign, 419
- Obesity in etiology of hypertrophic arthritis, 253
- Obturator nerve, 423
- Occupational cramp, 406
- Occupational therapy in chronic arthritis, 239
- Olecranon bursitis, 369
- Operative treatment in atrophic arthritis, 154
 bibliography on, 163
- Orthodoxybenzoic acid 152
- Osteitis deformans, 523

- Osteo-arthritis See Hypertrophic arthritis
 Osteo-arthritis See Hypertrophic arthritis
 Osteochondromata, 261
 Osteomalacia, 523
 Osteoporosis, 523
 Osteotomy, 177
 Oxoate, 152
- Pachymeningitis, chronic hypertrophic, 405
 Paget's disease, 523
 Pain in the shoulder girdle and arm
 neurofibroma as cause of, 404
 non arthritic conditions which may cause it, 404
 peripheral neuritis causing, 404
 pressure upon brachial plexus causing, 404
 Panniculitis, 361, 362
 Paraffin, hot, application in chronic arthritis, 233
 Paraplegia, 501
 Paravertebral injections, 438
 for hypertrophic arthritis, 286
 Parmanil, 148
 Pathogenesis of atrophic arthritis See Atrophic arthritis, pathogenesis of
 Pathology of atrophic arthritis See Atrophic arthritis, pathology of
 Periarthritis of the shoulder, 363
 bibliography on, 393
 clinical manifestations of, 389
 deformities in, 192
 etiology of, 388
 laboratory data in, 390
 pathology of, 389
 treatment of, 391
 drugs in, 391
 local, 391
 manipulation, 392
 physiotherapy, 391
 prevention of adhesions, 391
 roentgenotherapy, 391
 surgical exploration, 392
 systemic management, 391
 Peripheral circulation in atrophic arthritis, 51
 Peripheral neuritis, 404
 Peritendinitis, 361
 of shoulder, 388
 Peroneal nerve, 423
 Physical therapy in arthritis, 231, 304
 acetylcholine iontophoresis, 236
 baker for use in, 232, 233
 bibliography on, 246
 Physical therapy in arthritis—(Continued)
 compresses, hot, 234
 contrast baths, 234
 diathermy, 235
 exercise, 237
 heat, 232
 heliotherapy, 241
 histamine iontophoresis, 236
 inductothermy, 236
 low frequency currents, 239
 massage, 236
 occupational therapy, 239
 paraffin, hot, 233
 postural rehabilitation, 239
 radio-active substances, 241
 roentgenotherapy, 242
 short wave diathermy, 235
 systemic exposure to heat, 234
 x ray therapy, 242
 Physiotherapy, 231, 304, 435
 Pitkin maneuver, 441
 Pleurisy, 362
 Pleurodynia, 362
 Pneumococcal arthritis, 343
 bibliography on, 347
 classification of, 18
 treatment of, 343
 Polyarthritides, primary progressive See Atrophic arthritis
 Postural exercises, 435
 Postural low back strain, 453
 bibliography on, 454
 symptoms of, 454
 treatment of, 454
 Postural rehabilitation in chronic arthritis, 239
 Precipitating etiologic factor in atrophic arthritis, 35
 Pregnancy and atrophic arthritis, 106
 Prepatellar bursitis, 369
 Prevention of arthritis, 155
 Prevention and correction of deformities in chronic arthritis, 167
 arthrodesis for, 178
 arthroplasty in, 177
 Smith Petersen, for hip, 178, 209, 283
 vitalium mold, 178, 209, 283
 axioms with reference to, 180
 banjo-splints for, 173, 203, 207
 bibliography on, 181
 capsuloplasty in, 176
 capsulotomy in, 176
 casts, wedging of for, 173
 cervical spine, 183

- Prevention and correction of deformities in chronic arthritis—(Continued)
- elbow, 196
 - in the feet, 178, 222
 - fingers, 203
 - hand, 203
 - head, 183
 - hips, 208
 - illustrated guide to recognition of character, causes of, and means for, 183
 - knee, 210
 - lumbar spine, 191
 - manipulation, gradual manual, for, 172
 - manipulation under anesthesia for, 173
 - osteotomies in, 177
 - procedures employed for, 170
 - rest in, 171
 - shoulder, 192
 - skeletal traction for, 174
 - skin traction for, 172
 - Smith Petersen arthroplasty for hip, 178, 209, 283
 - spine cervical, 183
 - spine, lumbar, 191
 - spine, thoracic, 187
 - splinting of joints for, 169, 171
 - surgical procedures useful in, 174
 - synovectomy for, 176
 - tenotomy for, 178
 - thoracic spine, 187
 - traction, skeletal, for, 174
 - vitalium mold arthroplasty, 178, 209, 283
- Proliferative arthritis See Atrophic arthritis
- Pronation of feet, 222, 224
- Prostatitis in arthritis, 77
- Psoriasis arthritica See Psoriatic arthritis
- arthropathica See Psoriatic arthritis and atrophic arthritis, 105
- Psoriatic arthritis, 125
- bibliography on, 126
 - pathology of, 125
 - relation to atrophic arthritis, 125
 - treatment of, 125
- Psychalgias, 406
- Psychogenic makeup of candidate for arthritis, 28
- Psychotherapy in atrophic arthritis, 154
- Pyarthrosis See Acute suppurative arthritis
- Pyogenic arthritis See Acute suppurative arthritis
- of spine, 458
- Pyriformis muscle, 418, 470, 472, 479
- affections of, 472, 479
 - bibliography, 480
 - myotomy for treatment of, 480
 - treatment of, 480
 - anatomic relationships of, 479
 - myotomy of, 480
- Queckenstedt test, 503
- Quincke's edema, 365
- Radicular irritation, 399
- Radiculitis, 399
- secondary to hypertrophic arthritis of cervical spine, 399
- Radiculomyelitis, 406
- Radioactive substances in chronic arthritis, therapy with, 241
- Radiohumeral bursitis, 370
- Raynaud's disease, 84
- Renal infection in arthritis, 77
- Rest in treatment of atrophic arthritis, 131
- Reversed Queckenstedt test, 503
- Rheumatic arthritis, chronic See Atrophic arthritis
- Rheumatic disease, chronic, 3
- disabling effect of, 3
 - economic burden imposed by, 4
 - economic loss caused by, 3
 - facilities for treatment of, 5
 - hospital care of, practicability of, 9
 - hospital facilities for the care of, 7
 - incidence of, 3
 - invalidism caused by, 3
 - national health survey on, 3
 - physicians for the care of, training of, 10
 - post graduate teaching centers for, 11
 - socio economic aspects of, 3
 - bibliography on, 11
 - United States public health survey on, 3
- Rheumatic fever, 82, 85, 107, 291, 321, 328, 330
- abdominal manifestations in, 297
 - Aschoff nodule in, 293
 - atrophic arthritis, relationship between, 294
 - bibliography on, 305
 - blood count in, 299
 - blood cultures in, 299
 - cardiac damage in, 296
 - cardiac involvement in, 298
 - formol gel reaction indicating, 299
 - signs of, 298
 - classification of, 18

Rheumatic fever—(Continued)

- clinical manifestations of, 295
- differential diagnosis of, 301
- electrocardiographic changes in, 298, 300
- endocarditis in, 298, 299
- erythema multiforme, 297
- erythema nodosum in, 297
- etiology of, 291
- familial susceptibility in, 292
- foci of infection and, 294
- formol gel reaction in, 299
- growing pains in, 296
- hyperergy in, 293
- laboratory aids in diagnosis of, 299
 - bibliography on, 307
- nutritional disturbances in etiology of, 292
- pathogenesis of, 293
- pathology of, 293
- pleurisy in, 298
- pneumonia in, 298
- roentgenographic findings in, 300
- salicylates in treatment of, 302
- sedimentation rate of erythrocytes in, 299
- serologic findings in, 299
- skin manifestations in, 297
- subcutaneous nodules in, 294
- sulfanilamide in, 303
 - bibliography on, 307
- susceptibility in, familial, 292
- symptoms of, 295
- synovial fluid, cultures of, 299
- treatment of, 301
 - acetylsalicylic acid in, 303
 - aminopyrine in, 303
 - bed rest in, 302
 - blood transfusions in, 303
 - climatotherapy, 304
 - diet in, 302
 - focal infection, eradication of, 303
 - iron in, 303
 - local therapy in, 302
 - medicinal, 302
 - nonspecific protein shock in, 304
 - physical therapy in, 304
 - preventive measures in, 304
 - salicylates in, 302
 - sodium salicylate in, 303
 - sulfanilamide in, 303
 - vaccines in, 304
 - vitamins in, 302
- vaccine therapy in, 304
 - bibliography on, 307

Rheumatic fever—(Continued)

- vitamin C metabolism in, 292
 - bibliography on, 307
- vitamin deficiency in, 292
- Rheumatic headache, 275, 362, 403
 - treatment of, 283
- Rheumatic ophthalmia, 23
- Rheumatic state See Rheumatic fever
- Rheumatic subcutaneous nodules, 85, 294
- Rheumatism, chronic, responsibility of
 - family physician in control of, 7
 - muscular, 361, 366
 - requiring morphine, 151, 519
 - tuberculous See Tuberculous rheumatism
- Rheumatoid arthritis See Atrophic arthritis
- Rheumatoid arthritis of the spine See Atrophic arthritis of the spine
- Rheumatoid spondylitis. See Atrophic arthritis of the spine
- Roentgenotherapy in chronic arthritis, 242, 365, 439
- Sacralization of fifth lumbar vertebra, 464
- Sacro-iliac articulations. See Sacro-iliac joints
- Sacro-iliac disease, 417
 - bibliography on, 477
 - causes of, 472
 - diagnosis of, 474
 - Gaenslen's sign in, 473
 - roentgenographic findings in, 474
 - symptoms of, 473
- Sacro-iliac joints, 423, 470
 - abnormalities of, 470
 - as cause of low back and sciatic pain, 470
 - incidence of, 470
 - treatment of, 475
 - anatomic relations of, 427, 470
 - subluxation of, 471
 - surgical fusion of, 476
 - tuberculosis of, 471
- Sacro-iliac slips, 440, 474
- Sacro-iliac subluxation, 474 501
- Salicylates, 151, 302
- Scalenus anticus muscle, 394
- Scalenus anticus syndrome, 394 See Cervical rib and the scalenus anticus syndrome
- Scarlatinal rheumatism See Arthritis of scarlet fever
- Scarlet fever, arthritis of See Arthritis of scarlet fever
- Schmorl nodule, 496

- Sciatic nerve, 423, 479
 injection of, 438
 segmental distribution of, 423, 424
 Sciatic pain See Low back and sciatic pain
 Sciatica, 394, 404 See also Low back and sciatic pain
 Sedimentation of erythrocytes, 92, 149
 299, 320
 in atrophic arthritis, 149
 in gouty arthritis, 320
 Sedimentation test in atrophic arthritis, 92
 technique of, 93
 Seminal vesiculitis in arthritis, 77
 Senescent arthritis See Hypertrophic arthritis
 Septic arthritis, 107
 Serologic studies in atrophic arthritis, 39
 Serum protein concentration in atrophic arthritis, 135
 Serum sickness, arthritis of See Arthritis of serum sickness
 Shoes, 225
 Short wave diathermy in chronic arthritis 235
 Shoulder
 deformities of, 192
 frozen, 383
 in hypertrophic arthritis, 273
 involvement in atrophic arthritis, 88
 manipulation of, 196
 pain in, 377
 pain in, nerve involvement as cause of, 399
 periarthritis of, 388
 deformities in, 192
 stiff, painful, 388
 Sinus infection in atrophic arthritis, 76
 Skeletal traction, 217, 219
 Smith Petersen arthroplasty employing vitallium mold, 178, 209, 283
 Snapping finger, 367
 Sodium auro thiomalate, 147
 dosage of, 148
 precautions in administration of, 149
 Sodium salicylate, 151, 302
 Sodik, 345
 Specific arthritis, non infectious, 351
 Specific infectious arthritis, 107, 347
 classification of, 18
 Spina bifida occulta, 463
 Spinal cord, compression of, 405
 Spine
 arthritis of, 457
 atrophic, 457
 bibliography on, 461
 Spine—(Continued)
 arthritis of—(Continued)
 hypertrophic, 273, 403, 457
 pyogenic, 458
 specific types of, 458
 symptoms of, 458
 syphilitic, 458
 treatment of, 458
 tuberculous, 337, 458
 atrophic arthritis of See Atrophic arthritis of the spine
 carcinomatous infiltration in, 405
 cervical, in atrophic arthritis, 88
 deformities, arthritic, of, 183
 hypertrophic arthritis of, 273, 403, 457
 in hypertrophic arthritis, 273, 403, 457
 treatment of, 283
 congenital anomalies of, 463
 horizontal sacrum, 464
 sacralization of fifth lumbar vertebra, 464
 spina bifida occulta, 463
 disease in vertebrae of, 405
 dorsal, in hypertrophic arthritis, 276
 fractures of, 405, 522
 hypertrophic arthritis of, treatment of, 283
 involvement in hypertrophic arthritis, 273, 403, 457
 treatment of, 283
 lumbar, deformities in, 191
 in hypertrophic arthritis, 276
 sprains of, 522
 syphilis of, 523
 thoracic, deformities in, 187
 tuberculosis of, 337, 458
 in typhoid, 346
 Splint, aeroplane, 195
 castex, 171, 198, 202
 Splints, banjo, 203, 207
 castex, 171, 198, 202
 cellulose compound, 171, 198, 202
 cock up, for wrist, 202
 Spondylitis ankylopoietica See Atrophic arthritis of the spine
 syphilitic, 523
 tuberculous, 337
 typhoid, 346
 Spondylolisthesis, 464
 clinical manifestations of, 465
 differential diagnosis of, 467
 roentgenographic findings in, 466
 treatment of, 467
 Spondylose rhizomélique See Atrophic arthritis of the spine